ARCHIVES of OPHTHALMOLOGY

VOLUME 13 (old series Volume 70) MAY, 1935 NUMBER 5

THE FORMATION OF PAPILLEDEMA

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Papilledema, defined as edema of the optic disk, produced by increased intracranial pressure, is one of the most important symptoms of intracranial as well as of ocular pathologic change. The great importance of this symptom was recognized by the great Albrecht von Graefe. Ophthalmologists have gradually learned to distinguish between the actual conditions of choked disk, optic neuritis and pseudoneuritis, and have become familiar with the aspect of this symptomatic appearance of the disk when originating from causes other than increased intracranial pressure. As causes of the appearance of papilledema one considers immediate pressure on the optic nerve and, under certain conditions, diminished intra-ocular pressure.

It is known that papilledema is really only an edematous swelling of the intra-ocular portion of the optic nerve without any of the inflammatory changes which can and do appear in later stages of the condition. As long as the alterations in the disk are limited to edema only, one finds but slight functional disturbances, appearing in the enlargement of the blindspot. Other functional disturbances occurring in the early period of papilledema are not due to the swelling of the disk, but must be attributed to the condition causing the papilledema itself. Naturally, tumors of the brain, abscess of the brain, hydrocephalus and similar diseases can affect the optic pathways in different and most severe manners and simultaneously cause both visual trouble and papilledema. The oculist must see to early and efficacious treatment of the fundamental disorder, so as to prevent secondary changes in the optic nerve which threaten to destroy vision by atrophy of the nerve. Suppression of the cause of papilledema in later periods of the condition frequently fails to prevent progress of the atrophy, which ultimately leads to blindness. Notwithstanding sound knowledge of the causes and symptoms of papilledema, there exists no certainty as to the pathogenesis of the phenomenon, i. e., the mechanism of its formation. There are a number of theories concerning the formation of papilledema, which will pass in review presently, but none of them bears thorough criticism.

Read before the Medical Society of the District of Columbia at Washington, Oct. 3, 1934.

THEORIES OF THE MECHANISM OF PAPILLEDEMA

Von Graefe believed that increased intracranial pressure acted by compression of the cavernous sinus, as a consequence of which venous stasis led to swelling of the disk. Similar ideas had already occurred to Türck. Von Graefe, however, demonstrated that a pathologic condition located in any part of the brain may cause a dangerous state leading to blindness by calling forth changes in a remote place, viz., the disk. Von Graefe drew attention to the fact that the anatomic conditions in the disk are favorable to the production of stasis in this very place. Sesemann,[1] however, demonstrated that obstruction of the cavernous sinus does not produce stasis in the central vein, because of the connection between the superior ophthalmic vein and the inferior ophthalmic and anterior facial veins. Thrombosis of the cavernous sinus does not necessarily produce papilledema. Its appearance seems generally to point to the presence of some complication. Knape, Deyl and Dupuy-Dutemps [2] modified the theory in presuming that the compression of the central vein takes place at the entrance of the vein into the optic nerve or in the intervaginal space. Von Hippel [3] expressed the belief that the venous obstruction is secondary to the edema. Paton and Holmes, however, were of the opinion that the venous obstruction is primary.

The "transport theory" of Schmidt-Rimpler and Manz [4] is based on the fact that there is open communication between the subdural and subarachnoidal spaces of the cranial cavity and the optic nerve. Increase of intracranial pressure raises the pressure in the intervaginal spaces of the optic nerve and leads to their dilatation, which is witnessed in from 60 to 70 per cent of cases of papilledema. This dilatation occurs perhaps even oftener. Elschnig,[5] however, pointed out that in bilateral papilledema the dilatation of the subdural space can be present on one and absent on the other side. It has been claimed that the cerebrospinal fluid can act by compression of the optic nerve superficially or by dilating the perivascular lymphatic spaces in the optic nerve; that it can prevent the circulation of lymph from the globe toward the cranial cavity, dilate the perivascular lymphatic spaces, compress the vessels, reach the disk

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1. Sesemann, E., cited in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 1, Leipzig, W. Engelmann, 1898-1900, vol. 2, p. 154.

2. Dupuy-Dutemps, Louis: Pathogénie de la stase papillaire dans les affections intra-cyaniennes, Thèse de Paris, 1900.

3. von Hippel, E.: Deutsche med. Wchnschr. **51**:1225, 1925; Krankheiten des Sehnerven, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, W. Engelmann, 1923, vol. 7, pt. 2 B.

4. Manz, W.: Arch. f. Ophth. **16**:265, 1870; Thesis, Heidelberg, 1887, p. 18.

5. Elschnig, A.: Arch. f. Ophth. **41**:179, 1895.

and push the cribriform plate into the globe. Schieck [6] supported this theory by anatomic examination in fresh cases of papilledema.

Jackson, Benedikt [7] and Dor [8] believed that edema of the optic nerve is the consequence of vascular irritation on a nervous basis.

Parinaud, Ulrich, Sourdille, Klauber [9] and Liebrecht claimed that edema of the brain spreads to the optic nerve and reaches the disk, in this way causing the edema.

Gowers, Leber, Deutschmann and Elschnig were of the opinion that the cerebrospinal fluid in tumor of the brain contains toxins and, reaching the optic nerve through the subdural and subarachnoidal spaces, causes inflammatory changes.

Behr [10] claimed the existence of a system of lymphatic spaces throughout the optic nerve, through which liquid from the vitreous continuously flows toward the cranial cavity. This lymphatic system is perfectly separated from the subdural and the perivascular spaces. The only communication between these spaces exists in the place of entrance of the central vessels into the optic nerve. Liquid can, however, flow only from the optic nerve into the submeningeal spaces, not in the inverse direction. High intracranial pressure presses two folds of the dura near the cranial end of the optic canal against the optic nerve. These folds act as ventils and hinder the circulation of the lymph from the globe to the cranial cavity. There appear first edema of the nerve fibers and subsequently venous stasis.

The theory that papilledema is inflammatory must be rejected, as there are no inflammatory signs in fresh cases of papilledema. Experiments leading to the formation of papilledema by an increase of intracranial pressure and the regression and perfect disappearance of papilledema and venous stasis in the fundus under the influence of decompression oppose the theory of irritation and of propagation of edema of the brain.

The experiments of Marckwort, who by ligature or compression of the optic nerves produced ischemia of the disk and not edema, seemed to refute von Graefe's theory. During the last two decades, the theories of Schieck and Behr were in the foreground, and arguments were produced in favor of both as well as arguments to refute them. It is,

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6. Schieck, F.: Die Genese der Stauungspapille, Wiesbaden, J. Bergmann, 1910; Arch. f. Ophth. **78**:113 and 157, 1911; Thesis, Heidelberg, 1910, p. 179; 1918, p. 281.

7. Benedikt, M.: Allg. Wien. med. Ztg. **13**:21, 1868.

8. Dor: Nouvelle theorie de la stase papillaire, Thèse de Nancy, 1898.

9. Klauber, E.: Klin. Monatsbl. f. Augenh. **60**:504 and 764, 1918.

10. Behr, C.: Arch. f. Ophth. **83**:519, 1912; **89**:265, 1915; **101**:165, 1919-1920; Klin. Monatsbl. f. Augenh. **49**:672, 1911; **55**:193, 1915; **57**:465, 1918; **59**: 241, 1917; Ztschr. f. Augenh. **71**:275 (July) 1930.

however, difficult to imagine that the intra-ocular pressure of from 18 to 24 mm. of mercury should be able to press liquid from the eye through the entire length of the optic nerve as far as the cranial cavity, finding an obstacle at the entrance into the cranial cavity, if the intracranial pressure is considerably increased. Intra-ocular tension does not rise in consequence of increased intracranial pressure. On the contrary, the difference of pressure in the eye and in the skull is very important in these conditions. The cribriform plate is pressed toward the globe in cases of high intracranial pressure. If, however, the intracranial pressure is lower than the ocular tension, the cribriform plate is pushed backward, as Noiszewski [11] proved in his experiments.

STUDIES OF RELATION OF RETINAL ARTERIAL TO RETINAL VENOUS PRESSURE

The point of issue of the idea which I should like to present to you concerns measurements of venous and arterial blood pressure in the retinal vessels in normal and in diverse pathologic conditions. Bailliart [12] and after him a number of investigators have studied these questions. Attention was chiefly turned, however, to the arterial pressure and its relations to pathologic conditions. Bailliart also gave valuable information as to the behavior of venous pressure, and Baurmann [13] followed him in this line of work. These authors found relations between venous pressure in the retina and intracranial pressure. Dr. Sobański,[14] of my clinic, first checked up their results, systematically taking the pressure in normal persons and in persons with pathologic conditions. His investigations gave the height of arterial and venous systolic and diastolic pressure in the retina in relation to the general blood pressure at different ages. As results of his investigations he gives:

1.—Venous pulsation in the retina, synchronic with the carotid pulse, whether it is spontaneous or provoked by pressure on the globe, appears at the moment when the intra-ocular tension becomes equal to the pressure in the retinal veins. In this respect it behaves in the same way as arterial pulsation. The venous pulsation is caused by the arterial pulsation which reaches the veins through the capillaries (theory of Van Tright).

2.—At the age of 7 to 15 years, venous retinal pressure is generally at its minimum below the intra-ocular tension and amounts to from 16

11. Noiszewski, C.: Okulistyka, 1921, Warsaw; Polskagaz. lek., 1922, nos. 17-20.

12. Bailliart, P.: Ann. d'ocul. **81**:267, 1918; **167**:321, 1928; La circulation retinienne a l'état normal et l'état pathologique, Paris, Gaston Doin & Cie, 1923.

13. Baurmann, M.: Theses, Heidelberg, 1925, p. 53; 1926, p. 96; 1928; Arch. f. Ophth. **118**:118, 1927; Klin. Monatsbl. f. Augenh. **83**:115, 1929; Klin. Wchnschr. **11**:2071 (Dec. 10) 1932.

14. Sobański and Szczeniowski: Klin. oczna **4**:360, 1933.

to 22 mm.; its maximum, to 28 mm. Between 15 and 40 years, the minimum is 20 mm.; the maximum, 34 mm. Beyond the age of 40, the minimum is 23 mm.; the maximum, 36 mm.

3.—The minimum and maximum of arterial retinal pressure at the age of from 7 to 15 years are 40 and 68 mm.; from 15 to 75 years, the minimum is from 48 to 56 mm. and the maximum from 80 to 90 mm. The proportion of the minimum of venous to the minimum of arterial pressure lies between 1 : 1.9 and 1 : 3. The relation of the arterial pressure in the retina to the general blood pressure (arteria brachialis) lies for the minimum between 1 : 1.4 and 1 : 1.5, and for the maximum, between 1 : 1.3 to 1 : 1.6.

STUDIES OF RELATION OF INTRACRANIAL PRESSURE TO RETINAL ARTERIAL AND VENOUS PRESSURE

Careful examination of ninety-three patients in the neurologic clinic, in whom immediately after the pressure in the retinal vessels was measured, the tension of the cerebrospinal fluid was measured by lumbar puncture, led to the following results:

1.—The venous retinal pressure stands in intimate relation to the intracranial pressure.

2.—The height of the venous pressure defined by the appearance of venous pulsation gives a sufficiently accurate measure of the intracranial pressure, especially in cases with increased intracranial pressure. If the intracranial pressure is below 250 mm. of water, the error does not exceed 4 mm. of mercury; if the intracranial pressure is above 250 mm. of water, the error is below 2.5 mm. of mercury.

3.—In all cases of increased intracranial pressure, the diastolic arterial pressure was increased, whether papilledema was present or not. The increase of diastolic arterial pressure can be of diagnostic significance only when the general blood pressure is normal and the vascular system does not show any changes. Therefore its application is limited, and it does not allow one to define the height of the intracranial pressure even approximately. The measuring of the venous retinal pressure with the ophthalmodynamometer of Bailliart, Baurmann or Sobański is, if correctly executed, a sufficiently exact method for the definition of intracranial pressure.

Procedure.—After dilatation of the pupil by euphthalmine hydrochloride and superficial anesthesia of the globe, the intra-ocular tension is taken. Under control of a luminous ophthalmoscope, the veins in the disk are observed in the direct image, the ophthalmodynamometer is applied over the insertion of the external rectus, and pressure is exercised on it until the first contraction of the vein becomes visible. This is the procedure in the absence of spontaneous venous pulsation. Should there be spontaneous venous pulsation, which means that the minimum

venous pressure lies below the intra-ocular tension, the latter must be lowered by pressure on the globe until, on relieving this pressure, the veins show no pulsation. This condition obtained and the intra-ocular tension having been taken again, the same procedure is applied as described before. In 1 per cent of the cases there is no spontaneous venous pulsation, and it cannot be elicited by pressure on the globe. This means that the venous pressure lies below the intra-ocular tension. By releasing the pressure exercised on the globe, venous pulsation can appear and then the venous pressure can be measured. For measurements, only venous pulsation synchronic with the carotid pulse can be utilized. The level of minimum venous pressure in the retina, expressed in millimeters of mercury multiplied by 10, gives the intracranial pressure in millimeters of water. It is judicious to repeat the measurement three times and take the average. As a last act, the intra-ocular tension is taken once more for control purposes.

The described procedure is sufficiently exact for practical purposes and allows one to take the intracranial pressure at different times of the day. By means of this investigation one can control the therapeutic action of treatment. As the procedure is perfectly innocuous, it ought to be applied in all cases in which one wants information as to intracranial pressure and considers that the examination of the cerebrospinal fluid is not necessary. It is especially important in the case of a tumor in the posterior cranial depression, in which lumbar puncture can be eminently dangerous.

The examination of ninety-three cases has proved that the method is really reliable.

Increase of intracranial pressure almost immediately causes increase of retinal venous pressure. This was proved by pressure exercised on the trephined places in cases of intracranial hypertension in which there had been surgical intervention. Immediately after the beginning of pressure, the retinal veins showed dilatation and tortuosity. The venous tension rose in a few minutes from 20 to 25 mm. of mercury to between 32 and 35 mm. The disks became hyperemic, and vessels not visible before appeared to the observer.

EFFECTS OF EXPERIMENTAL VARIATION IN THE PROPORTION OF VENOUS TO ARTERIAL RETINAL DIASTOLIC BLOOD PRESSURE

Compression of the jugular veins leads to increase of the intracranial pressure (Queckenstedt's sign) and after twenty seconds makes the venous retinal pressure rise. Consequently, every increase of intracranial pressure causes increase of retinal venous pressure. If, in cases of increased intracranial pressure, papilledema appears, one always finds a disturbance of the proportion of diastolic arterial to venous retinal pressure. In cases of papilledema, this proportion rises above 1 : 1.5; i. e.; venous tension has risen to such an extent that the difference between it and arterial pressure is small. In cases of high intracranial pressure without papilledema, the relation of venous to arterial pressure

remains normal; i. e., it varies between 1 : 1.9 and 1 : 3. The level of retinal arterial pressure is high.

These clinical observations illustrate the relation between intracranial and intra-ocular pressure. Experimental research led a step further.

As mentioned in the beginning, von Graefe's theory was especially refuted by experiments of Schultén,[15] Wahlfors and Marckwort, who compressed the optic nerve immediately behind the globe and caused complete anemia of the fundus without signs of stasis. In consequence of these observations, all influence of retinal vessels on the formation of papilledema was denied. Sobánski [14] repeated these experiments. When the optic nerve was completely compressed immediately behind the globe, complete ischemia of the retina was obtained. Both artery and vein were compressed in this case. When, however, the nerve was compressed only so far as to suppress venous circulation without interrupting

Fig. 1.—Experimental papilledema in a dog.

arterial circulation, matters took a completely different aspect. Constant ophthalmoscopic control showed rapid dilatation and tortuosity of the retinal veins; after three minutes the disk became hyperemic; its limits began to be indistinct. Six minutes after the beginning of the experiment, the disk was distinctly prominent, and fifteen minutes after the beginning the difference of level amounted to from 2.5 to 3 diopters. A microscopic section of an eye subjected to this experiment in comparison with one of the control eyes illustrates these facts distinctly (figs. 1 and 2). If pressure was exercised on the optic nerve in the posterior part not containing central vessels, these changes did not appear. Similar results were obtained by Behr in a case of orbital tumor, in which before the exenteration of the orbit, its content was ligated at the apex of the orbit. Papilledema appeared.

15. Schultén, M. W.: Arch. f. Ophth. (pt. 3) **30**:1, 1884.

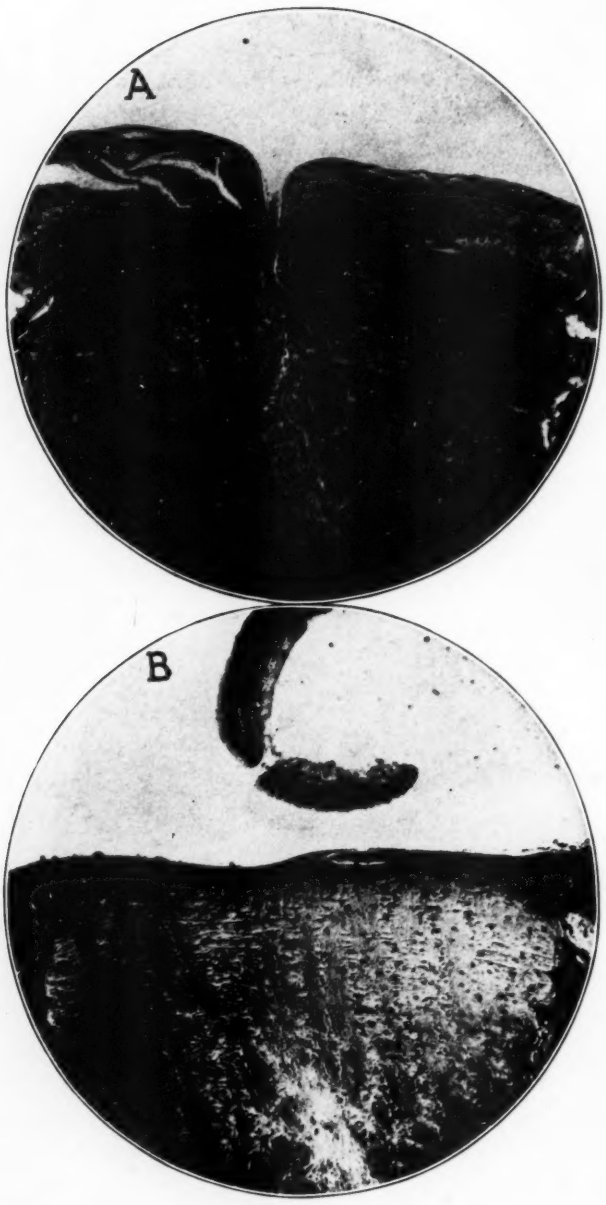

Fig. 2.—*A*, microscopic section showing experimental papilledema in a dog; *B*, section through the control eye of the same dog.

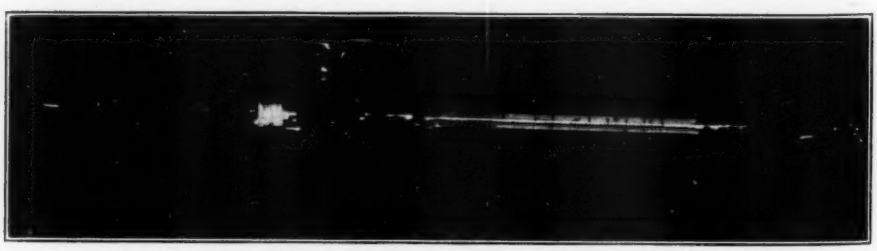

Fig. 3.—Ophthalmodynamometer of Dr. Sobański.

LOCATION OF THE VENOUS OBSTRUCTION CAUSING ALTERATION IN THE RATIO OF VENOUS TO ARTERIAL RETINAL DIASTOLIC PRESSURE

The clinical observations and the results of experiments tend to prove that obstruction to venous circulation in the optic nerve plays a great part in the formation of papilledema. There remains to ascertain where the obstruction to venous circulation is located.

Anatomic facts make it improbable that obstruction occurs at the inosculation of the central vein and the cavernous sinus. Experimental investigations of Lamach, Claude and Targowla proved that the blood pressure in the sinuses is partly independent of the intracranial pressure. The venous communications in the orbit will in some cases allow the blood to seek its way instead into the cavernous sinus and, if the pressure in the latter should be increased, into the facial veins.

There is no doubt that the pressure in the intervaginal spaces of the optic nerve is increased if the intracranial pressure rises. This has repeatedly been proved experimentally (Quincke;[16] Schnitzler and Albert[17]). Wahlfoors showed that ligature of the optic nerve behind the entrance of the central vessels into the nerve prevents the formation of papilledema when intracranial pressure is raised. The accumulation of cerebrospinal fluid between the sheaths of the optic nerve is a necessary condition for the formation of papilledema. The syndrome of Foster Kennedy[18] is proof of this. The cases observed by Schultz-Zehden,[19] Kubik[20] and Mariotti[21] are further proof.

MECHANISM OF PAPILLEDEMA

All observations and experimental facts support the idea that increase of diastolic venous pressure in the retina and its relation to the diastolic arterial pressure in the retina are decisive for the formation of papilledema. If arterial tension in the retina is low, even a slight increase of venous pressure caused by high intracranial pressure produces papilledema. In the presence of low arterial pressure, a venous pressure of from 28 to 30 mm. of mercury leads to hyperemia of the disk with indistinct limits; in slightly higher pressure (35 mm. of mercury) the characteristic image of papilledema appears. If, on the contrary, diastolic arterial pressure is high, a venous pressure of 35 mm. causes no hyperemia of the disk; hyperemia appears when the pressure rises to from

16. Quincke: Cytowany przez Lebera.

17. Schnitzler and Albert, cited by Leber in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, W. Engelmann, 1901, vol. 2, p. 484.

18. Kennedy, F.: Am. J. M. Sc. **142**:355, 1911.

19. Schultz-Zehden, P.: Klin. Monatsbl. f. Augenh. **43**:153, 1905.

20. Kubik, J.: Klin. Monatsbl. f. Augenh. **71**:353, 1923.

21. Mariotti, C.: Boll. d'ocul. **8**:1111, 1929.

40 to 45 mm., the picture of papilledema developing at a still higher pressure. In one case, intracranial pressure amounted to 450 mm. of water, and venous retinal pressure was 50 mm. of mercury, yet the disk was only slightly hyperemic and its limits somewhat blurred. Arterial pressure in the retina was 130 systolic and 85 diastolic; the proportion of venous to arterial minimum pressure was 1 : 1.7. In all cases in which high intracranial pressure did not lead to papilledema, the aforesaid proportion lay between 1 : 1.7 and 1 : 2.6. The alteration of this proportion to between 1 : 1.7 and 1 : 1.5 always leads to hyperemia of the disk and to blurring of its margins. Alteration of the proportion beyond 1 : 1.5 always leads to papilledema.

An interesting point is the relation of the appearance of the disk in cases of low intra-ocular tension. In such cases one can observe papilledema *ex vacuo*. Such cases have been known for a long time. Kyrieleis [22] studied these facts in 1929. Very illustrative was a case of glaucoma in which operation was performed on both eyes by cyclodialysis at my clinic. The tension of the right eye for a certain length of time remained between 14 and 20 mm., whereas that of the left eye declined to between 6 and 8 mm. Before the operation, both disks had been similar, i. e., slightly pale with physiologic cupping. After the operation, the appearance of the right disk remained unaltered; the left disk showed swelling up to 2.5 diopters and small hemorrhages on its surface. Venous pressure in both retinas was 25 mm.; arterial tension in the right eye was 55 systolic and 85 diastolic and in the left eye, 38 systolic and 70 diastolic. The proportion of the venous to the arterial minimum pressure was 1 : 2.2 in the right eye and 1 : 1.5 in the left. The general blood pressure was 150 systolic and 95 diastolic. When, after some time, the intra-ocular tension of the left eye rose to 36 mm., the prominence of the disk disappeared, leaving only hyperemia and slight blurring of the disk margins. An acute attack of glaucoma raised the tension to 70 mm. After iridectomy, the tension fell to 2 mm., and the disk showed an elevation of 2 diopters.

Paton and Holmes showed that if there should be any difference in the tension of both eyes, papilledema is more marked in the eye with the lower intra-ocular tension. Such cases have also been observed at my clinic. It is known that general blood pressure has scarcely any influence on intracranial and intra-ocular pressure, and also that it does not influence retinal venous pressure. Retinal venous pressure, however, is about twice the height of intra-orbital venous pressure. From these facts one can deduce that the pressure of the cerebrospinal fluid in the intervaginal spaces of the optic nerve keeps the venous pressure of the retina on a comparatively high level.

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22. Kyrieleis, W.: Arch. f. Ophth. **121**:560, 1929.

One could consider as an objection to the proffered theory of the formation of papilledema the fact that in thrombosis of the central vein of the retina one does not see papilledema. This absence of papilledema in this condition is, however, only apparent. Examination with the binocular ophthalmoscope in cases of thrombosis of the central retinal vein shows an excessive thickening of the retina by edema, which is so great that the likewise strongly edematous disk does not protrude above the level of the retina. Papilledema is consequently present also in this condition but can not be recognized without the binocular ophthalmoscope of Gullstrand.

The proportion of the retinal venous to the arterial pressure is of importance not only for the formation but also for the further development of papilledema. In low arterial pressure, even a comparatively slight increase of intracranial and consequently of retinal venous pressure leads to great prominence of the disk, pronounced venous stasis, retinal hemorrhages and an early appearance of secondary inflammatory symptoms which modify the ophthalmoscopic picture and make diagnosis difficult. In such cases early decompression is especially necessary in order to prevent rapid loss of vision.

Diseases of the blood—chlorosis and polycythemia—lead to papilledema on account of high intracranial pressure, which has been found in both conditions. In orbital tumors, compression of the central vein will lead to rise of pressure in the retinal veins.

The enumerated facts enable one to explain the formation of papilledema on the basis of a comparatively simple mechanism. Low diastolic blood pressure in the retinal arteries with normal intra-ocular tension and simultaneous rise of venous retinal pressure are the main conditions leading to formation of papilledema. My associates and I are busy seeking further proofs for the theory expounded. I hope and am certain that experimental control by many investigators observing closely the same technic will corroborate our statements.

We are at work. No attainment is ever final. The more knowledge that is acquired on papilledema the greater will be the number of sufferers relieved.

ARACHNODACTYLY

(DYSTROPHIA MESODERMALIS CONGENITA, TYPUS MARFANIS; MARFAN'S SYNDROME; DOLICHOSTENOMELIA)

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BROOKLYN

In 1896, Marfan[1] called the attention of physicians to a peculiar familial condition featured by very long and thin bones. The syndrome was so clearcut that other cases were soon recognized and studied. Prior to 1932, about eighty-four cases had been reported, but with the present recognition of associated conditions of the eyes and heart it is probable that many more instances will be found by ophthalmologists and pedologists.

The entire bony system is affected, but the changes are especially noticeable in the hands and feet and the spinal column. The hands are very long and narrow, with delicate fingers, a feature which has suggested the name now commonly used—arachnodactyly. The arms and legs are long and slender and the feet so much longer than normal that the patient has great difficulty in getting shoes to fit. The spinal column and the muscles are not strong enough to sustain the weight imposed and in all the cases reported here there was more or less spinal curvature. The skull and teeth are long and narrow. The bony palate is narrow and the arch high. The absence of subcutaneous fat exaggerates the slimness and height of the patient and gives an impression of frailty.

The roentgenogram shows no abnormalities in the bones other than those mentioned, except that spina bifida occulta was found in the two patients of my group who were examined roentgenologically. Dr. Rendich and Dr. Bell, who made the examinations for me, stated that this peculiarity is rather common among all classes of patients and would have no significance unless it occurred regularly.

About one half of the patients whose cases had already been reported had congenital heart disease, but the details of this feature were not available. None of the patients in the present series had this condition. Another feature found in about the same ratio is congenital miosis that even atropine cannot overcome. The feature in these cases of greatest interest to the ophthalmologist is congenital bilateral dislocation of the lens. About one half of the total number of patients examined, and five of the six patients I have seen, have shown this malformation.

Read at the Seventieth Annual Meeting of the American Ophthalmological Society, Lucerne, Canada, July 11, 1934.

1. Marfan, A.: Bull. et mém. Soc. méd. d. hôp. de Paris **13**:220, 1896.

Oculists should have the condition in mind whenever a child is found with congenital miosis or bilateral dislocation of both lenses. In the ophthalmic clinics general examination of the patient showing either

Fig. 1.—*A*, father's hand; *B*, Louis de V.'s hand, showing arachnodactyly.

Fig. 2.—Roentgenogram of the thorax of Louis de V. The bones are very thin and delicate but are otherwise normal. There are no signs of heart disease.

defect should be required, and the pedologist should be as interested as the ophthalmologist.

Some authors think that this disease is due to an endocrine disturbance, but it is the fashion now either to attribute exerything to the lack

of a particular vitamin or to cast the blame on one or more of the ductless glands. The official name of the condition, "dystrophia mesodermalis congenita, typus Marfanis," implies that all these peculiarities are to be attributed to defective development of the mesoderm, since all the tissues affected, except the dilator fibers of the iris and the suspensory ligament of the lens, arise from this layer. It will be seen later that these two tissues may not be involved.

Fig. 3.—Father and mother of the de V. family. The mother has the characteristic bony defects of the disease, but the eyes are normal.

REPORT OF CASES

In October, 1932, Louis De V., aged 9 years, was admitted to Dr. Beery's clinic at the Brooklyn Eye and Ear Hospital. With Dr. Beery's permission I am able to report not only five cases from a family of eight but also another case in a family of two children and one parent. Vision in each eye was 10/200 without correction; this improved to 10/100 with a +8 sph. Retinoscopy revealed +7.00 diopters with high astigmia at axis 120 in one eye and +7.00 diopters with a similar high astigmia at axis 60 in the other. The pupils dilated about one third of the

usual degree after the instillation of atropine. The lenses were dislocated upward and outward, leaving a thin crescent of clear, black pupil at the lower, inner part of the pupil. The lenses were clouded with many fine dots scattered everywhere, which shut out a view of the fundi. Because of the small area of pupil uncovered and the thickness of the lens, the fundus could not be studied. The fibers of the suspensory ligaments were clear, strong and very long, as though the lens had been pulled to one side rather than as though it was lacking in support. The fibers of the zonula showed fine red-brown dots, and similar dots studded the freely movable vitreous strands that could be seen. The patient had marked lordosis

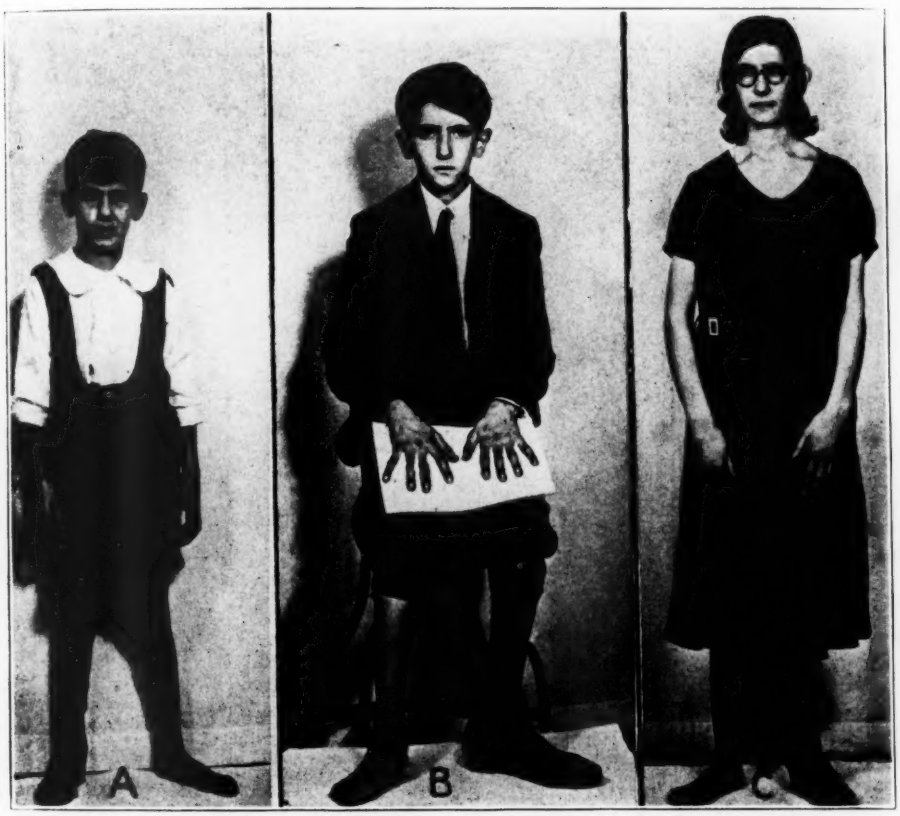

Fig. 4.—*A*, Joseph de V., aged 5; *B*, Louis de V., aged 9, and *C*, Philomena de V., aged 13. A fourth child, a girl of 18, would not allow her picture to be taken.

and the characteristic features seen in such cases. The long, narrow skull with the characteristic facial expression, due in part to the poor vision, and the long, thin, narrow teeth make it possible to pick out a patient with a typical case at a glance. At the age of 9 the patient had a foot as long as mine, and his father complained bitterly of the trouble occasioned whenever a change of shoes was necessary.

There are six children in this family; four—two boys and two girls—have the disease, and two—one boy and one girl—have escaped. The father is not affected, but the mother has all the skeletal features shown by the diseased children; her

eyes, however, are normal. The youngest boy was 4½ years of age when seen. He had very poor vision—about 10/60 in one eye and 10/200 in the other—but it was difficult to do much with him as he was nervous, fidgety and undisciplined. His eyes were like his brother's, with bluish-gray lenses, upward and outward dislocation and long, strong, thick zonular fibers. He had marked spinal curvature and had been wearing a cast for several months. The picture gives the best description of him. No view of the fundus could be obtained.

The oldest sister, aged 22, did not have the disease. The next oldest sister, aged 18, had visual acuity of 10/40 in one eye and 10/30 in the other with a

Fig. 5.—Louis L., aged 8, whose vision was 10/200, with a sister, aged 15, who is normal.

+3.00 and a +2.50 sph., respectively. Atropine and homatropine had but slight effect. The fundi in this case could be seen and showed tapetoretinal degeneration, with dirty, pigmented spots and a decided tendency toward sclerosis of the choroidal vessels. The picture did not resemble that of retinitis pigmentosa. The next sister was 13 and had vision in one eye of 6/200, which was improved to 10/30 by a +5.00 sph., and vision in the other eye of 10/200, which was improved to 10/70 by a +4.00 sph. The lenses of these sisters were dislocated, like those of the brothers. In the eyes of the second sister, red-brown beads were attached to the fibers of the suspensory ligament and to some of the vitreous strands, and tapetoretinal degeneration similar to that in the other sister, with sclerosis of the choroidal vessels, was present. The mother is evidently the transmitter; her mother

is a frail little woman with none of the characteristics of the condition. The grandfather is dead, but was described as a tall man with long, thin fingers and feet much larger than those of his daughter. His eyes were reported to have given him no trouble. The family is pure Italian, as is the second family of present interest.

After the members of this family were seen and were examined, so far as was possible, another patient with a typical case was brought to Dr. Beery's clinic. He was 8 years of age and differed in no way from the other patients, except that he was much more restless and nervous, so that it was difficult to take the picture. Lordosis was marked; the feet were long, and the fingers typical. It would be hard to imagine two boys of families unknown to each other who resembled each other as closely as do these. The father and sister were normal. The mother had died of Hodgkin's disease after a long illness. From the description given of her, she was evidently the transmitter of the arachnodactyly. The grandparents were never in this country, and no details of them could be obtained. The boy had vision of 10/200, which could not be improved. The lenses were bluish gray and could not be seen through; both were dislocated outward and a little upward. The zonular fibers were clear and strong, but no details of the fundus could be seen.

COMMENT

Objection has been made to the term mesodermal dystrophy because the zonule and the dilating fibers of the iris do not originate from the mesoderm. Kubik removed the lens from a woman of 25. She had 20 diopters of near sight and 6 diopters of far sight, using the lens-free pupil. On the side toward which the lens was dislocated was a coloboma. The lens weighed 170 mg. Bakkur studied two lenses removed after prolapse into the anterior chamber. The age of the patient or patients or whether the lenses came from one or two persons was not stated. The weight and diameter of the first lens, however, were similar to those of the lens of a new-born infant. The thickness was increased, but there was defective development for the period of late intra-uterine life. There were remains of embryonic tissue on the posterior capsule, with a fissure at the pole. The fibers of the lens were irregularly arranged, with the zonular fibers attached well toward the posterior margin of the edge of the lens. The second lens showed changes dating farther back than those of the first. The heaping up of nuclei of the lens fibers on the posterior portion of the lens and the other changes spoke for overdevelopment of the anterior portion of the lens. It is suggested that the unyielding pupil is caused not by lack of dilator fibers but by the rigid framework of the iris.

The cases reported to date furnish the following details: Males are affected as often as females. The disease is familial and heritable. Mild and atypical cases may escape notice, but the disease is much more common than is supposed. While the thinness and length of the bones has been emphasized, the weak musculature is just as much to blame for the spinal curvature as is the frail spinal column.

Only two Wassermann tests could be made in this group of six cases, but the results were negative, and there was no sign of syphilis in any of the patients, unless the tapetoretinal degeneration noted in the two sisters can be considered the result of inherited syphilis without other indications. These patients were difficult to handle, and one was fortunate in being able to obtain as many data as were secured.

LECTURES ON MOTOR ANOMALIES OF THE EYES

IV. FUNCTIONAL NEUROSES: ETIOLOGY, PROGNOSIS AND TREATMENT OF OCULAR PARALYSIS

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Disturbances of the motility of the eyes due to functional neuroses and hysteria require consideration not only because of their importance in the making of a differential diagnosis, but also in order to clear up the many misunderstandings and contradictions found in the interpretations that have been given by various authors. Whether or not purely functional paralysis of ocular muscles occurs remains to this day a moot point. Even Uhthoff,[1] while refusing, like so many ophthalmologists and neurologists, to accept hysteria as a cause of the paralysis, has, strange to say, referred in his writings to "publications which affirm beyond doubt the exceptional occurrence of hysterical paralyses of even individual muscles of the eye."

Nevertheless, I think that one is not only entitled but indeed obliged to doubt the reliability of the observations reported as instances of purely hysterical paralysis. To the best of my knowledge, the investigations made in those cases were not exact and exhaustive, the results were not interpreted correctly or the length of time available for the observation was not sufficient so that a disease organic in nature but complicated with hysterical symptoms could not be excluded as a possible cause of the paralysis. The dictum of Mauthner that the frequency of hysterical disturbances of ocular muscles is directly proportional to the possibility of producing at will the condition concerned is still valid today. Accordingly, spastic disorders of ocular muscles are met most frequently in cases of hysteria, since the corresponding innervation is governed by the will and the spastic movements can be learned by exercise and initiated by suggestion. Sometimes blepharospasm is the only hysterical stigma, and it is often caused by trauma or disease of the eyes or of their adnexa. Convergence spasm also occurs frequently in hysteria, sometimes as its only symptom, but generally associated with spasm of accommodation and contraction of the pupils. The inconstancy of the angle of squint and the prompt success of suggestive treatment prove the functional nature of the convergence spasm, notwithstanding the fact that the cause could be an anomaly of organic origin.

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1. Uhthoff, W., in Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1915, vol. 11, pt. 2 B.

During the World War I examined a soldier who nine months before had been wounded near the interior angle of the eye by a piece of splintered shell. He complained of diplopia, poor vision, headache and vertigo. I found that he had spastic ptosis, spasm of convergence and accommodation, intense miosis, and continuous tremor of the head. The poor vision, due to spasm of accommodation, could be improved by concave glasses; when one eye was covered it became immediately normal without any glass. A thorough examination of the muscles of the eyes revealed the presence of paralysis of both depressor muscles of the left eye due to the trauma. When asked concerning the diplopia, the patient told me that he had been greatly disturbed at first by the double vision, the images being at different levels. Endeavoring to overcome the trouble, he found some relief when he made a strong convergence effort, because the distance between the two images became greater while simultaneously they became indistinct.

The case is interesting because it shows the development of the characteristic ocular symptoms of a functional neurosis, based on paralysis of organic origin. The symptoms of paralysis were hidden by the strong convergence spasm so that they could be revealed only by an exceptionally careful examination. Until this was done, every kind of treatment had been, of course, fruitless. However, the disappearance of the spasms could be immediately effected, albeit only temporarily, by putting an opaque glass before one eye to remove the troublesome diplopia. The patient was definitely cured as soon as binocular single vision was restored by advancement of the paralyzed inferior rectus muscle.

In some cases of hysteria conjugate deviation of the eyes has been observed. That it is not caused by an associated paralysis is always easy to prove.

Nystagmus of hysterical origin is rare and never the only symptom of hysteria; it is almost always connected with spasm of the orbicularis and convergence muscles. The nystagmic movements are exceedingly fine and rapid, much more rapid than in any other kind of nystagmus.

Hysterical associated paralyses of the movements of the eyes have been repeatedly described, especially during and after the war; they were caused by hardships and emotional excitement or by severe nervous shock. At first the symptoms may be similar to those in cases of so-called pseudo-ophthalmoplegia: inability to move the eyes at a word of command and in some cases to follow the moved object of fixation; on the other hand, the fixation was maintained when the patient's head was passively rotated not only by a sudden jerk but also quite slowly. But at times spontaneous movements of the eyes could be observed when the patient was left to himself or was talking with other patients or reading a paper. The true functional nature of the paralysis is, however, proved only by the success of suggestive treatment.

That the manifestations of convergence or divergence paralysis are sometimes based on a functional neurosis, and how this can be proved, has already been discussed.

Some cases of hysterical divergence spasm, combined with other hysterical symptoms, have been described by Kehrer and other authors. In my opinion a patient with this type of condition has an exophoria which becomes manifest intermittently, especially during attacks of blepharospasm, in which the eyes have a tendency to diverge on account of their elevated position.

I have never seen as a true functional disturbance paralysis of individual eye muscles or unilateral ophthalmoplegia, and in the publications on this subject I have not found a description of any case which convinced me that the supposition of a functional origin of the paralysis was correct. Either the proof of the paralytic character of the related symptoms was equivocal, so that they could as well have been due to nonparetic anomalies, or the patient was not kept under observation long enough to exclude organic nervous disease, which of course may be complicated by hysterical symptoms. Only one-sided, nonspastic, hysterical ptosis definitely occurs. This is not strange, as a normal person also can train himself to relax one of the elevators of the upper lids completely. One-sided, spastic (pseudo-)ptosis is much more frequently described as a hysterical sign, but it must be emphasized that in such cases also the starting point could well be an organic lesion.

Several years ago I was asked to give an opinion on a patient suffering from various troubles which he attributed to contusion of the head sustained in an automobile accident. His physicians, not being able to find any evidence of an organic lesion, had made a diagnosis of traumatic neurosis. At last, after the case had been submitted to arbitration, I was asked about the nature of the blepharospasm in the right eye. After I had, with much trouble, induced the patient to open his right eye, I could not find anything abnormal in the eye itself, but when I investigated the motor apparatus I discovered a slight paresis of the superior rectus muscle of either eye. The patient had never complained of diplopia and did not even remember, when asked, ever having had it. But I was forced to admit that the blepharospasm was probably caused by the accident, perhaps by a hemorrhage within the nuclear region, and that the patient, although not conscious of seeing double, felt constrained by the blurred vision to keep one eye closed.

In recent publications, especially in Germany, another kind of hysterical disturbance has been discussed, namely, the so-called dissociation of movements of the eyes. It was described at first by Kunn as follows: The eyes are not held rigidly in a certain position as in real spasms, and they do not obey the patient's will but move about rather irregularly, one eye independent of the other as in a comatose condition. The supposition that the association of movements of the eyes can be temporarily suspended merely as a symptom of hysteria is a strong contradiction to all physiologic and clinical facts by which the general validity of Hering's so-called law of association of movements of the eyes is proved. Dissociated movements of the eyes occur in a comatose

state, in narcoses and during sleep, that is to say, in conditions in which the muscles of the eyes are governed neither by the will nor by the compulsion to fusion but may receive stimuli of subcortical origin. However, it is beyond one's volitional power to innervate either an individual eye muscle or the muscles of one eye alone, and the contrary assertion depends on a misunderstanding of Hering's law. The occurrence of one-sided or asymmetrical movements of the eyes by no means proves, as I emphasized in my first lecture, that these movements arise from asymmetrical innervations of the two eyes. If two innervations, one impelling the eyes to a parallel movement and the other impelling them in an opposite direction arrive at the eyes at the same time, the result must be an asymmetrical or one-sided movement in spite of the fact that both eyes are equally influenced by the same impulses. The symptoms adduced as proof of dissociation cannot, on critical examination, be accepted as such. They could be due to heterophoria becoming manifest intermittently according as the patient is attentive and interested in the test object or tired and listless, or to organic disease complicated with hysteria. Both could not be excluded in any given case. Since some of these patients were unable to fix an object in spite of normal mobility and sufficient vision of the eyes, I am inclined to assume that there was a lack of voluntary impulses because of an inhibited cortical function, as is the case in the hypnotic state. What is most characteristic of hysterical disturbances of ocular movements is their inconstancy and amenability to treatment by suggestion. In any case of hysteria complicated by apparently unusual motor anomalies of the eyes one must take into consideration the various possibilities of their origin, as just discussed, particularly the coincidence of simple heterophoria with hysterical spasm and organic lesions.

ETIOLOGY OF OCULAR PARALYSIS

The etiology of ocular paralysis as the basis for treatment cannot be discussed here in detail, but I should like to refer to some results of my own researches as far as those results differ from the views recorded in textbooks. An interesting point worth mentioning is the indubitable increase in the percentage of cases of paralysis of the trochlear nerve, which I was able to ascertain by the comparison of my material collected within the last twenty-five years with that of the preceding years. This increase cannot in any way be attributed to an improvement in the methods of investigation, since all the cases included in these statistics have been examined solely by myself. Up to the year 1908 the number of cases of trochlear nerve paralysis amounted to 10 per cent of all cases of paralysis, not quite half as many as the cases of paralysis of the abducens nerve. From that time to last year the percentage of cases of trochlear nerve paralysis was about 20, and that of abducens nerve paralysis the

same as before, about 25 per cent. The striking increase in the number of cases of trochlear nerve paralysis is due, I am sure, to the introduction of Killian's operation and of similar radical operations on the frontal sinus. These operations came into vogue between the years 1903 and 1908. In these operations after the periosteum is cut through the trochlear nerve recedes into the orbit. If the operation is finished without refixing the nerve to its original attachment by exact periosteal sutures, the function of the superior oblique muscle is weakened. In the majority of cases this weakness lessens gradually and disappears within a few weeks, but in some cases it becomes permanent and displays the typical manifestations of paralysis of the trochlear nerve.

In the eighty cases of trochlear nerve paralysis which I have seen within the last nine years in my clinic, no less than fifteen had been caused by operations on the frontal sinus. A traumatic origin was found in 15 per cent of all my cases of paralysis. Syphilis and metasyphilis constitute the most frequent cause, and in statistics published years ago the percentage of cases due to these conditions was from 50 to 60. In 1906 I found that 55 per cent of cases belonged in this group, nearly the same as the percentage found by Sauvineau. This percentage has decreased considerably since then. Less than 30 per cent of the cases of paralysis collected within the last ten years were of syphilitic or metasyphilitic origin. In all the cases the blood and spinal fluid were examined. The indubitable decrease in the occurrence of ocular paralysis due to syphilis and metasyphilis is to be attributed in all probability to modern therapy of syphilis.

Next in frequency are the cases of paralysis of congenital origin and paralysis caused by epidemic encephalitis (14 per cent each). Unfortunately it is impossible to enter into the particulars of the interesting and extremely multifarious disturbances of the oculomotor apparatus which are encountered in cases of epidemic encephalitis and of postencephalitic paralysis and spasm. I must also omit discussion of certain well known etiologic factors, such as intracranial diseases, anomalies of the blood vessels and acute infectious diseases. Among the paralyses of toxic origin, those due to spinal anesthetics, especially benzoyl-dimethyl-amino-ethylpropanol, procaine hydrochloride or tropacocaine, have been long known and numerous; they have been mostly paralyses of the sixth nerve, less frequently of the trochlear nerve and only exceptionally of the third nerve. As a rule the paralysis arises a few days after the injection and takes some weeks to disappear. Opinions differ as to whether the nuclei or the nerves are damaged and how they are damaged by the poison. In the course of time the frequency of the appearance of these paralyses has gradually decreased. During the last nine years in my clinic among 600 cases of ocular paralysis there has not been a single case caused by a spinal anesthetic.

Because of its extreme rareness I shall mention two cases of paralysis of the abducens nerve due to severe loss of blood.

One patient had been wounded by a file and had lost an enormous quantity of blood. When he was discharged from the hospital, he was extremely anemic and complained of blurred vision and of seeing sparks before his eyes. I found, as the only cause of his troubles, a slight paralysis of the right abducens nerve. It diminished slowly and disappeared within a year.

From the second patient a considerable quantity of blood had been taken for the purpose of transfusing it into her husband, who suffered from leukemia. The following day she noticed diplopia, and according to her physician's report there was paralysis of both sixth nerves. When I saw her some months later, she displayed a convergence angle of 20 degrees, increasing neither in looking to the right nor to the left. Improvement was slow; she was finally cured after thirteen months.

Finally, it should be noted that in spite of the most careful investigation the etiology of ocular paralysis still remains obscure in a comparatively high percentage of cases, in my material in 15 per cent.

PROGNOSIS OF OCULAR PARALYSIS

A few comments may be permitted with respect to the prognosis in cases of ocular paralysis. Spontaneous recovery occurred in 38 per cent of my cases, the percentage being, however, extremely different for the various forms of paralysis. The highest percentage (about 57 per cent) was found in cases of paralysis of the trochlear nerve, nearly 50 per cent in paralysis of the abducens nerve, but only 28 per cent in paralysis of the third nerve, in ophthalmoplegia and in associated paralyses. The reason for these differences is easy to understand. The majority of cases of paralysis of the fourth and of the sixth nerve are caused by either a trauma or a tiny nuclear hemorrhage which may be reabsorbed within a short time, whereas in the majority of the other paralyses the lesion is more serious and of greater extent, and more than 50 per cent of them are due to syphilis or metasyphilis—all factors most unfavorable as regards prognosis. Among the cases of spontaneous recovery from the paralysis, the cases with an obscure etiology constitute the highest percentage. In the majority of these cases the paralysis may be of toxic or infectious origin or may be caused by tiny nuclear hemorrhages. Sometimes such paralyses disappear within a few days or even hours, as suddenly as they appeared.

As to how soon operation for the correction of paralytic deviations is advisable, it is important to know that if six months have elapsed since the inception of the paralysis the possibility of spontaneous recovery is extremely meager. I remember patients with traumatic paralysis who presented a constant condition, especially a constant restriction of mobility and a constant angle of squint, for six months and only then

began to improve, and finally recovered completely. Therefore I never operate for paralytic deviation until at least six months have passed.

TREATMENT OF OCULAR PARALYSIS

As for the treatment of ocular paralysis, the causal indication must be considered primarily. The often surprising results of antisyphilitic therapy in cases of paralysis caused by cerebral syphilis are well known, but even in cases in which the nature of the paralysis cannot be detected a cure is effected by means of diaphoretics, mercury, iodine and other medicaments. Patients suffering from hemorrhages due to disturbances of the circulatory apparatus must take laxatives, have their diet regulated and remain in bed for several weeks.

The local treatment during the first stage is only palliative. Occlusion of the paralyzed eye to remove intolerable diplopia is often unavoidable, but it must be limited to the shortest possible time because the exclusion of the fusion tendency aids the development of secondary contracture and does not engender a compensatory innervation.

As long as the angle of squint does not remain constant in the different directions of vision, prism spectacles do not help at all, not to mention the fact that prisms stronger than 4 or 5 degrees cannot as a rule be endured and also that prisms cannot correct a meridian deviation of the eye, i. e., disclination or conclination. Galvanic treatment, though much used, is in my opinion of use only as a suggestive measure, for because of the danger of damaging the retina the current must be so weak that it does not produce a contraction of the normal muscles of the eye.

If with nonoperative treatment no success or only partial improvement has been obtained, operative treatment must be considered in order to correct the position and, if possible, the mobility of the eyes, as well as to restore binocular single vision at least in the middle part of the field of fixation; a good result would at the same time remove the anomalous position of the head. There are few operations in ophthalmology as satisfactory as the operations under discussion provided the oculist has carefully chosen the type of operation best suited to the particular case and provided, if the case is so complicated that the desired result cannot possibly be obtained by a single operation, the patient has sufficient faith and perseverance to undergo several operations.

The paralytic deviation of one eye must be corrected by increasing the efficiency of the paralyzed muscle. This result can be obtained by advancing or shortening the muscle, even if the paralysis is incurable. Assuming that an eye muscle as an agonist must overcome the resistance of its antagonist, oculists formerly thought that the function of a paralyzed muscle might be improved by weakening the antagonist. How-

ever, Sherrington has shown that the lengthening of an antagonist in the normal act of vision is not to be understood as a passive stretching produced by the contraction of the agonist, but is due to an active relaxation which is accomplished even if the agonist does not function at all. It is now realized that it is impossible to improve the function of a paralyzed eye muscle by weakening its antagonist.

It is principally owing to Landolt's persistent propaganda that oculists have gradually ceased to employ tenotomy in every case of deviation, whatever its origin. It is known that though paralytic squint may be improved by tenotomizing the antagonist of the paralyzed muscle, this advantage is more than offset by the addition of postoperative insufficiency of a normal muscle to the paralysis of its antagonist: The result in a case of paralysis of the abducens nerve would be that in the whole field of fixation possibly only one point could be seen single and homonymous diplopia would occur in looking to one side and crossed diplopia in looking to the other side of that point. In absolutely rejecting tenotomy Landolt has indeed overshot the mark. A long-standing, permanent paralytic deviation of high degree due to strong secondary contracture of the antagonist cannot be corrected solely by advancement or shortening of the paralyzed muscle. Landolt recommended that if the result of advancement is not satisfactory, for instance in a case of paralysis of the left abducens nerve, the right external rectus muscle should be advanced in preference to tenotomy of the left internal rectus, which would involve the risk of insufficiency of this muscle. If, in the example mentioned, the secondary contracture of the left internal rectus muscle is not corrected and the normal muscle balance of the right eye is destroyed by advancing the external rectus muscle, in the position of rest both eyes will be directed to the right so that the patient, in order to look at an object in front of him and to see it single, will be obliged to turn his head to the left. A habitual anomalous position of the head is certain to result from Landolt's procedure. In the cases under discussion the secondary contracture of the internal rectus muscle must be remedied by tenotomy or better still by recession of the muscle. There is no objection to this, since its excessive function is not of the least use to the patient and must be reduced to its normal measure and there will be no disadvantage in the act of seeing.

Without discussing in detail the various operations on the muscles of the eyes I should like to say a few words about my guiding principles for operations for strengthening and weakening the eye muscles. Strengthening can be effected either by an advancement or by a shortening operation. Personally, I prefer a combination of advancement with resection of a larger or smaller portion of the tendon according to the amount of deviation and to the degree of paralysis. In no case is it

certain how many degrees of deviation will be definitively corrected by an advancement or a shortening operation. The results obtained by identical operations in similar cases differ within wide limits, and to discuss the reasons would lead me too far away from the main subject. Claude Worth, though admitting that it is impossible to estimate beforehand the results that will be obtained by operation, has maintained in his well known monograph on squint that the immediate effect of his advancement operation is also the permanent result and that therefore no overcorrection is necessary. On this point I do not agree with Worth. The immediate effect of advancement, as well as of every shortening method, diminishes in the course of time, and I have always been satisfied if I have obtained a considerable overcorrection as an immediate result of operation.

As I have said before, it is wrong to refuse on principle to perform the muscle-weakening operations. The contrary opinion, advocated lately also by von Liebermann,[2] is based on the assertion that the effect of such operations is always incalculable and always produces a form of paralysis of the weakened muscle. Reference is further made to the disfiguration due to the postoperative exophthalmos and the recession of the caruncle. Indeed, these considerations are true enough for a simple tenotomy, but not for a recession operation safeguarded by a suture by which the separated tendon is prevented from receding farther back than is desirable. Only in certain cases, to be discussed later, is it necessary to insert the suture into the sclera. I prefer as a rule to pass one needle of the double-armed suture through the tendon before I cut it and then through the peripheral edge of the divided conjunctiva; the other needle is passed through the conjunctiva near the corneal margin. Then I make a loose loop of the ligature, which can be pulled tight or still further loosened or removed on the following day or later according to circumstances. So I am able to modify the result to a certain degree, in any case preventing an unwelcome overcorrection and cosmetic disfigurement. To the champions of advancement and shortening operations it may be replied that if the correction of a deviation is to be obtained in every case and solely by these operations, disadvantages almost as unwelcome as those with which the weakening operations are reproached must often be anticipated. The enophthalmos which is always associated with narrowing of the lid fissure resulting from an excessive advancement causes a cosmetic disfiguration as ugly as the exophthalmos and the receding of the caruncle following an unguarded tenotomy. Moreover, excessive advancement of a paralyzed muscle will restrict the function of its normal antagonist as much as an unguarded tenotomy of the latter.

2. von Liebermann, L.: Ueber Muskelvorlagerung, Ztschr. f. Augenh. **66**:209 (Oct.) 1928.

From these facts, which have been verified by many observations, I have derived my own principles of procedure in typical cases of paralytic squint. I shall begin with a consideration of treatment of paralysis of the abducens nerve.

Advancement and shortening of a paretic external rectus muscle is the operation of choice in uncomplicated cases. This will correct a deviation up to 15 degrees and restore binocular single vision in the greater part of the field of fixation. The abduction of the paretic eye can become nearly normal.

If the muscle is totally paralyzed, its function cannot be restored and one must be content to improve the position of the paralyzed eye and of the head. The prognosis is much better if the loss of function is caused not by paralysis but by an unguarded tenotomy, as will be seen in the photographs of a patient who was tenotomized fifty years ago because of convergent squint (fig. 1). The internal rectus muscles, the tendons of which had receded behind the equator, were unable to move the eye

Fig. 1.—Total loss of function of both internal rectus muscles due to an unguarded tenotomy. There is deficient adduction of each eye in levoversion and dextroversion (*A* and *B*). After advancement and shortening of the internal rectus muscles the visual lines are parallel (*C*) and the adduction of both eyes has become normal (*D* and *E*).

inward beyond the midline. The advancement of these muscles not only produced parallelism of the visual lines when the patient looked straight forward but also restored an adduction of normal extent. Why the results obtained by one and the same operation differ so fundamentally according to whether the loss of function is caused by paralysis or by the receding of the separated tendon beyond the equator is easy to understand. In the latter case the muscle, if innervated, contracts normally, the effect remaining latent only as long as the muscle is attached on the posterior half of the bulbus, whereas the paralyzed muscle does not obtain any innervation even after the operation.

Transplantation of the temporal halves of the superior and inferior rectus muscles to the site of the paralyzed external rectus was recommended first by Hummelsheim. It has not been used so much in Germany as in this country, as far as I can judge from recent publications.

Fairly good improvements are reported but, as far as I can see, only when the transplantation is combined with tenotomy of the internal rectus muscle. I have performed transplantation in several cases, but I was not so satisfied with the results as to be induced by them to abandon the other and simpler procedures in the cases under discussion. It may be possible to obtain by transplantation a decrease of the deviation and even a certain amount of power of abduction, but the patient will not be able to see objects in front of him single in the normal position of the head, and he will not give up the habit of anomalous rotation of the head toward the paralyzed side.

In long-standing cases of permanent paralysis I make the *modus procedendi* dependent on the behavior of the antagonistic internal rectus muscle. My main endeavor is to obtain a comfortable binocular single vision in the middle part of the field of fixation, so that the normal position of the head is regained. If in spite of total paralysis the deviation is below 15 degrees the desired result is obtainable by simple advance-

Fig. 2.—Paralysis of the right external rectus muscle with extreme contracture of the internal rectus muscle. There is extreme deviation of the paralyzed eye while the other eye is looking straight forward (*A*). Excessive adduction of the right eye occurs if the patient is looking to the left (*B*). The right cornea remains in nearly the same adduction if the patient looks to the right (*C*). After operation the visual lines are parallel in the primary position of the eyes and there is binocular single vision of distant objects (*D*). The right eye remains in the primary position if the left eye is looking to the right (*E*). Adduction of the right eye is restricted a little in looking to the left (*F*).

ment and resection of the paralyzed muscle; but in the majority of cases the deviation is of greater degree owing to secondary contracture of the internal rectus muscle. Since in these cases the adduction is abnormally increased in addition to advancement of the external rectus muscle, the internal rectus should be receded in the manner already described so as to bring its function down just to normal.

In a third group extreme contracture of the internal rectus muscle holds the visual line nearly immovably at the internal canthus (fig. 2). In my cases the paralysis was caused by fracture of the base of the skull.

Because of disturbing diplopia, the first patient, an automobile racer, had been unable to race for more than two years. He had consulted many oculists and had been told that his condition was inoperable. For such a case the usual proce-

dure would not have been sufficient. The man could be made fit to resume his work only by providing him with binocular single vision in the primary position of his head and eyes. To attain this the function of the enormously contracted internal rectus muscle had to be sacrificed almost completely. I resected 15 mm. of this muscle and advanced the external rectus muscle as far as possible in order to prevent a disfiguring exophthalmos. The photographs show the result: In looking straight forward with the head in its normal position the visual lines are parallel and are kept in this position without any effort. Although the side-to-side movements of the right eye are almost abolished, so that the patient has homonymous or crossed diplopia, according as he looks to the right or to the left, he is happy because he can do his racing as successfully as before his accident. He is not disturbed by diplopia except in near work, for which he uses spectacles with one opaque glass. More than five years have passed since the operation, and only a few weeks ago he wrote to me about success in racing which he had obtained.

I have repeated the described procedure in several similar cases with the same good result. It is surprising, how quickly—usually within a few days—the patient learns to avoid diplopia by turning his head as a substitute for the lost muscular functions, and in such an unaffected manner that persons with whom he talks do not notice the absence of the eye movements.

Sometimes the paralyzed eye is used permanently for fixation because of amblyopia of the nonparalyzed eye. As a rule considerable contracture develops in the internal rectus muscle of this eye. In such cases this muscle may be weakened without danger, since there is no diplopia or anomalous position of the head to be feared. Otherwise the weakening of the internal rectus muscle of the paralyzed eye without simultaneous advancement of the external rectus muscle would give rise to an ugly exophthalmos. If the same cosmetic result can be obtained by a simple as by a complicated operation, of course the former must be chosen.

I shall discuss now the operative treatment of inveterate paralysis of the trochlear nerve. Comparing the function of the external rectus muscle with that of the superior oblique, it is obvious that paralytic deviation in trochlear nerve paralysis cannot be corrected so easily as that in paralysis of the abducens nerve, since in trochlear nerve paralysis three deviation components must be considered: a vertical, a rotating and a lateral component, the last, however, being of subordinate importance.

Before one can decide whether and how a vertical deviation can be corrected by operation, the following questions must be answered: 1. Does the vertical deviation increase in looking up or in looking down? 2. Is it different in the right and left halves of the field of fixation? 3. Are the double images of contours parallel in the whole field of fixation or only in one part of it, being inclined toward each other in the other part? 4. Does tilting of the head around the sagittal axis without changing the direction of the visual lines influence the magnitude of the

vertical and the meridian deviation and if so in what manner? Only after having answered these questions by a careful examination is one in a position to know whether the deviation is due to nonparalytic heterophoria or to paralysis of one or several vertical motor nerves of the right or the left eye and what operative procedure to adopt.

I shall first consider the procedure in a typical case of trochlear nerve paralysis which presents both vertical and meridian deviations, increasing or decreasing in the various parts of the field of fixation, as was described in my second lecture,[3] I do not think it advisable to strengthen the function of a paralyzed superior oblique muscle because of its insertion in the posterior half of the bulb. The disturbed vertical balance of the eyes, i. e., the disturbed harmony of position and movements of the eyes, must be restored in another way. But tenotomy of the superior rectus muscle of the paralyzed eye is a wrong method and must be absolutely rejected, because it ignores the physiologic functions of the vertical motor nerves. The vertical deviation caused by trochlear nerve paralysis is indeed improved, even perhaps removed, by weakening of the superior rectus muscle, but only for a small middle part of the field of fixation. In the lower half, where the vertical deviation before the operation was greater, the effect of the operation is less than in the middle part, whereas in the upper part, where before the operation there was only a small vertical deviation or none at all, there may be even an overcorrection, that is, the contrary vertical deviation. If the patient is looking to the sound side, the vertical position of the paralyzed eye depending mainly on the oblique muscles, the vertical deviation due to paralysis of the trochlear nerve increases proportionately to the increasing adduction of the paralyzed eye, whereas it decreases in looking to the opposite side, because the vertical position of the abducted eye is mainly influenced by the vertical rectus muscles. Further, regarding the vertical deviation in the right and left halves of the field of fixation, tenotomy of the superior rectus muscle produces the maximum effect on the abducted eye and the minimum on the adducted eye—again an undesirable result. More unsatisfactory still is the effect of tenotomy of the superior rectus muscle on the meridian deviation in trochlear nerve paralysis. The paralytic deviation is increased by the operation because the rotating function of the superior rectus is the same as that of the superior oblique muscle, and the weakening of the former muscle likewise produces a deviation. The same disadvantage prejudices also the effect of an advancement of the inferior rectus muscle, as recommended by Landolt for the type of paralysis under discussion. The only advantage of this operation compared with tenotomy of the superior

3. Bielschowsky, A.: Lectures on Motor Anomalies of the Eyes: II. Paralysis of Individual Eye Muscles, Arch. Ophth. **13**:33 (Jan.) 1935.

rectus muscle is a better correction of the vertical deviation in the lower part of the field of fixation, but only in its middle part, as it is inadequate in the nasal quadrant of the lower half of the field of fixation.

Albrecht von Graefe's genius and his familiarity with the physiology of the muscles of the eye revealed the right way to remedy the after-effects of paralysis of the trochlear nerve. He was the first to perform tenotomy of the inferior rectus muscle of the sound eye. To Graefe's procedure, Stevens, Worth, Landolt and other eminent authors have objected that the weakening of the sound inferior rectus muscle would perhaps give a good result as regards the primary position of the eyes but would cause an overcorrection in looking down. This possibility must of course be considered, but it can be easily avoided if the inferior rectus muscle is weakened just to the extent needed to counterbalance the weakness of the paralyzed superior oblique muscle and not simply tenotomized. If this is successful, the vertical and the meridian deviation will disappear in nearly the whole field of fixation and the normal position of the head will be restored. This is easy to understand if one considers that the left superior oblique and the right inferior rectus muscle work in the same parts of the binocular field of fixation; both have the maximum influence as depressor muscles if the eyes are turned to the right and the minimum influence in the opposite side of the field of fixation; further, both these muscles incline the upper end of the vertical meridians to the right, so that the deviation caused by a paralysis of the left trochlear nerve is diminished or even transformed to parallelism when by weakening of the inferior rectus muscle the right vertical meridian is also inclined to the left, that is, to the same side as the left vertical meridian. The oblique position of both vertical meridians does not disturb the vision at all, provided they have become nearly parallel. This so-called compensatory operation has proved successful in many cases of paralysis of the trochlear nerve when the indispensable precautions for preventing an overcorrection have been taken. I always perform this operation in the following manner:

After the conjunctival incision the inferior rectus muscle is separated from the fascia and the fibers connecting the muscle with the conjunctiva. Then I put the needles of a double-armed suture from the posterior to the anterior side through the tendon and through the conjunctiva but through the latter farther back, near the region where it turns into the fornix or, if a high degree of vertical deviation must be compensated for, within the fornix. The ligature is tied loosely—not knotted twice—so that I am able, if necessary, to lessen or to intensify the effect of the operation on the following day. A careful suture of the conjunctival wound is necessary to prevent drooping of the lower lid, which looks ugly (fig. 3).

The procedure described, which I have used many times, has in no case resulted in a disturbing diplopia as a symptom of an overeffect. On the contrary, the disturbing double images of the lines, crossing each

other at an acute angle, which the patient had seen in reading, completely disappeared after the operation.

In the majority of cases of incurable paralysis of the trochlear nerve a contracture of the inferior oblique muscle prevents the decrease of the vertical deviation in looking upward as seen in typical cases, and finally the deviation becomes entirely independent of vertical movements, as I showed in the second lecture. In these cases recession of the inferior rectus muscle of the other eye can, to be sure, correct the deviation in the lower half of the field of fixation, but it is not sufficient for the correction in the upper half and in the horizontal plane. Since in most cases of this kind the function of the inferior oblique muscle is excessive, it may be weakened by severing it from its origin and resecting a few millimeters of it. There is no danger of obtaining an overeffect, as I was able to verify in about fifty cases. If a considerable amount of vertical deviation remains in the lower part of the field of fixation some

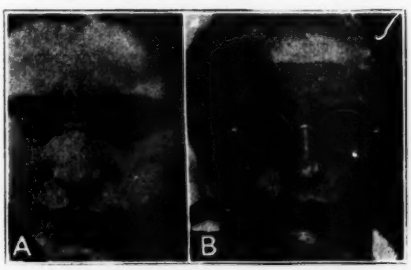

Fig. 3.—Compensatory weakening of the left inferior rectus muscle in a case of paralysis of the trochlear nerve. Drooping of the left lower lid occurs following a simple tenotomy of the inferior rectus muscle (*A*). After a careful suture of the conjunctival wound following recession of the inferior rectus muscle, there is no drooping of the lower lid (*B*).

weeks after the myectomy of the inferior oblique muscle, one may proceed to recession of the inferior rectus of the other eye, as previously described. The reverse order of succession of these two operations is less highly recommended, because in recession of the inferior rectus muscle a gradation of the effect is easier to bring about than in myectomy of the inferior oblique.

Similar to the cases under discussion are the cases of congenital anomalies, which I have described as *Ueberfunktion* of the inferior oblique muscle because of the lack of clues indicative of primary trochlear nerve paralysis. In almost all the congenital cases torticollis is displayed in earliest infancy as the first sign inducing the child's mother to consult a physician. About the orthopedic and surgical treatment which is frequently given in such cases, owing to the fact that the practitioner overlooks the ocular origin of the torticollis, I

have spoken in the second lecture. In most cases of this kind it is as surprising as it is gratifying to observe the child spontaneously straightening the formerly tilted head as soon as the ocular balance has been restored. This feature is easy to understand: The tilting of the head helps the patient avoid diplopia, which is caused by the excessive functioning of the inferior oblique muscle or of the insufficiency of its antagonist, as the case may be. That this anomaly is the only reason for the habitual tilting of the head is proved by the results of myectomy of the inferior oblique muscle followed if necessary by recession of the inferior rectus muscle of the other eye. As soon as the vertical deviation and the meridian divergence have been corrected and binocular single vision in the normal position of the head has been restored, the tilting of the head is abandoned. The photographs shown in figure 4 give an example of the result of the operations.

The congenital *Ueberfunktion* of one or both inferior oblique muscles is sometimes combined with a nonparalytic squint. If the former anomaly is not of too high a degree, it can be corrected by displacing

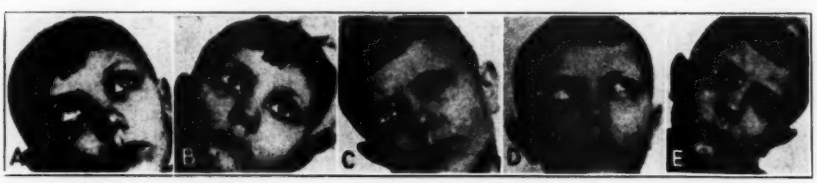

Fig. 4.—Congenital overfunction of the right inferior oblique muscle. Vertical deviation is increased *ad maximum* if the head is tilted toward the right side (*A*), and disappears if the head is tilted in the habitual way, i. e., to the left (*B*). After myectomy of the right inferior oblique muscle, there is no vertical deviation even if the head is tilted toward the right side (*C*) or if the patient is looking up and to the left (*D*). The habitual tilting of the head has been spontaneously given up after operation, since there is binocular single vision with the head straight as well as with it tilted to the right or left (*E*).

the external or internal rectus muscle, as the case may require, below the horizontal meridian of the bulb. As a rule the usual advancement and recession operations of the external and internal rectus muscles are sufficient also for the correction of the vertical deviation just discussed. Figure 5 shows a patient with a divergent squint of 25 degrees combined with an exceedingly high degree of so-called *Ueberfunktion* of the right inferior oblique muscle. Advancement of the internal and recession of the external rectus muscles of both eyes effected a perfect binocular single vision. There remained a fractional residue of the vertical deviation, manifesting itself only in maximum dextroversion. If in such a case the result with regard to the vertical deviation component is not adequate, a myectomy of the inferior oblique muscle is required.

In total paralysis of the oculomotor nerve as great an improvement as in the paralyses just discussed is of course not to be obtained, mainly because of the simultaneous deficiency of both the elevator muscles and of the inferior rectus. But in most cases one can obtain not only a cosmetic improvement but also binocular single vision at least in a small part of the field of fixation. The first and main task to be accomplished is the removal of the paralytic divergence by advancement of the internal rectus, combined, if necessary, with recession of the antagonist. When parallelism of the visual lines is obtained the patient learns quickly to find the position of the head in which a balance is struck between the vertical motor muscles and to replace the deficient vertical movements of the eyes by corresponding movements of the head.

The photographs of some of my patients with total paralysis of one or both oculomotor nerves may show the functionally perfect and cosmetically satisfactory results obtained by advancement of the internal and retroplacement of the external rectus muscles.

Fig. 5.—Overfunction of both the inferior oblique muscles combined with divergent squint. When the patient looks straight ahead the left eye is deviated outward (*A*). A levoversion impulse makes the right eye deviate upward (*B*), whereas dextroversion causes an upward deviation of the left eye (*C*). After advancement of both the internal rectus muscles and recession of both the external rectus muscles, combined with displacement of the four muscles below the horizontal meridian, the visual lines are parallel in the primary position (*D*) and in levoversion (*E*); a slight vertical deviation remains only in dextroversion (*F*).

The first patient (fig. 6) had tabes dorsalis and complete paralysis of the right oculomotor nerve. There was divergence of 40 degrees; the strongest impulse to look to the left could not move the right eye beyond the midline, but the lid fissure opened involuntarily—a pseudo-Graefe phenomenon, as described in my second lecture. Very interesting is the spontaneous disappearance of ptosis after restoration of binocular single vision in all cases in which the levator palpebrae has preserved some excitability, however little. The pseudo-Graefe phenomenon is still more striking after the operation when the patient is looking to the left, whereas the position of both upper lids is quite symmetrical in looking to the right. In spite of considerable recession of the right external rectus muscle, the abduction is not diminished at all, while a part of the adduction is regained by advancement of the internal rectus muscle.

The second patient (shown in fig. 7) fractured his skull six years before he was seen. Because of strong contracture of the superior oblique muscle, the right visual line remained below the horizontal plane, even when a maximum

elevating impulse was given. All the muscles of the right eye innervated by the third nerve were totally paralyzed, the pseudo-Graefe phenomenon being striking when the patient looked down. Therefore, from the beginning as good an operative result as that obtained in the previous case was not to be expected. However, binocular single vision of objects in front of the patient was obtained by advancement of the internal and recession of the external rectus muscle. Adduction was partially regained. Operation for ptosis was not advisable in consideration of the strong retraction of the upper lid in looking down and to the left.

In the third patient (fig. 8) the enormous divergence due to paralysis of both oculomotor nerves was corrected in the same way, and binocular single vision in looking straight forward was restored.

Fig. 6.—Paralysis of the right oculomotor nerve due to tabes dorsalis. The upper lid cannot be raised voluntarily (*A*) but is lifted involuntarily (pseudo-Graefe phenomenon) when a levoversion impulse is given (*B*) to which the paralyzed right internal rectus muscle does not react. After an advancement and shortening of the right internal rectus and a recession of the right external rectus, the visual lines are parallel in the primary position; there is no ptosis (*C*), and binocular single vision is restored. The adduction power of the right eye is considerably improved (*D*); abduction is normal in extent (*E*).

Fig. 7.—Paralysis of the right oculomotor nerve due to fracture of the base of the skull six years previously. The right eye cannot be moved in any direction except outward, and there is considerable ptosis (*A*). The maximum elevation impulse does not bring the right visual line up to the horizontal plane (*B*). Involuntary retraction of the right upper lid occurs as a result of the depression impulse (*C*). After advancement of the right internal and recession of the right external rectus muscle, there is binocular single vision in the primary position (*D*) and in the lower half of the field of fixation. A moderate adduction power of the right eye has been regained. Levoversion produces an automatic raising of the right upper lid (*E*).

The fourth patient (fig. 9) had paralysis of both oculomotor nerves due to congenital syphilis which caused a divergence of 60 degrees and forced him to turn his head habitually to the left, the left eye being permanently closed. The

photograph taken after the operation shows the satisfactory result, which enabled the patient to see singly with both eyes open the objects in front to him.

I should like to say a few words about operations for ptosis. For all patients with a normally functioning superior rectus muscle, the Motais operation, in which the middle third of the superior rectus muscle is substituted for the paralyzed levator palpebrae, should be the operation of choice. This procedure, based on the physiologic synergy of the movements of the eyeballs and the upper lids, gives highly satisfactory results; but it is advisable not to operate on young children

Fig. 8.—Total paralysis of both third nerves and an enormous secondary contracture of the external rectus muscles (*A*). After advancement and shortening of the internal rectus and recession of the external rectus muscles, there is parallelism of the visual lines and binocular single vision in the middle part of the field of fixation (*B*).

Fig. 9.—Total paralysis of both third nerves with enormous secondary contracture of both external rectus muscles caused by congenital syphilis. The patient habitually turns his head to the left in order to fix an object straight in front of him; the left eye is closed to avoid diplopia (*A*). In the primary position of the head, the paralytic deviation amounts to 60 degrees (*B*). After advancement and shortening of both internal muscles and recession of both of the external rectus muscles there is binocular single vision with the head in the normal position, the visual lines being parallel in looking straight forward (*C*).

because a child's superior rectus muscle is so small and delicate that the suture may easily cut through it. The photographs in figure 10 show a case of congenital bilateral ptosis. Only by a strong innervation of the frontalis muscle is the lid fissure opened a little. The successful result of the Motais operation, performed on both eyes, is seen in the photo-

graphs. The upper lids accompany the elevation as well as the depression of the eyes in the normal physiologic manner and are in the normal position also in looking straight forward. When the Motais operation is not applicable because of the complete absence of function of the superior rectus muscle, the operation recommended by von Blaskovics, is the best to adopt. In this operation a piece of the superior tarsus is removed and the levator muscle after the resection of its peripheral part is attached to the residue of the tarsus. The successful result is dem-

Fig. 10.—Congenital bilateral ptosis. The lid fissures are narrow (*A*); they can be opened a little more only by a strong innervation of the frontalis muscle and by an upward movement of the eyes (*B*). After a Motais operation there is binocular single vision in the primary position (*C*), in looking up (*D*) and in looking down (*E*). Both upper lids accompany the vertical movements of the eyeballs in the normal way. Particularly valuable is the complete relaxation of the elevator muscles of the upper lid in looking down; this cannot be obtained by the Hess operation.

Fig. 11.—Result of the von Blaskovics ptosis operation in a case in which the Motais operation was not applicable on account of a paralysis of the superior rectus muscles. The upper lids cover the upper halves of the corneas (*A*). After a von Blaskovics operation the lids assume a normal position (*B*). They can be relaxed just as completely when the eyes are looking down (*C*) as after a Motais operation.

onstrated by the photographs in figure 11. The Hess operation is still popular because of its simple technic, but it does not allow for the physiologic synergy of the lid and eye movements, since the upper lid, after it has been attached to the frontalis muscle, cannot be lowered when the eyes look down. Therefore I have abandoned this operation.

UNILATERAL EXOPHTHALMOS PRODUCED BY A MENINGIOMA OF THE MIDDLE CRANIAL FOSSA

REPORT OF A CASE

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AND
JOHN E. SCARFF, M.D.
NEW YORK

Slowly progressive unilateral exophthalmos associated with ophthalmoplegia, primary optic atrophy and hyperplastic thickening of the posterior orbital plate constitute a definite clinical entity. This syndrome is produced by a dural endothelioma or "meningioma" of the middle cranial fossa, arising from the region of the sphenoid ridge and greater wing of the sphenoid bone. This tumor has been variously described as meningioma of the sphenoid ridge, retro-orbital meningioma and meningioma *en plaque* (because of its peculiar flat form).

This condition was first clearly established as a definite clinicopathologic entity by Cushing [1] in 1922. Ten years later Elsberg, Hare and Dyke [2] reported a series of similar cases and Dr. Elsberg again discussed this subject at the meeting of the Section of Ophthalmology of the New York Academy of Medicine, May 21, 1934. Recently Alpers and Groff [3] of the neurosurgical clinic of Frazier wrote about "meningeal fibroblastomas" arising from the sphenoid ridge. In his textbook on intracranial surgery Bailey [4] discussed this condition. But, on the whole, it may be fairly said that familiarity with this syndrome is chiefly limited to the larger neurosurgical clinics and that among general

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Read before the Section of Ophthalmology of the New York Academy of Medicine, Oct. 15, 1934.

From the Department of Ophthalmology and the Department of Surgery of the New York Post-Graduate Medical School and Hospital, Columbia University.

1. Cushing, H.: The Cranial Hyperostoses Produced by Meningeal Endotheliomas, Arch. Neurol. & Psychiat. **8**:139 (Aug.) 1922.

2. Elsberg, C. A.; Hare, C. C., and Dyke, C. G.: Unilateral Exophthalmos in Intracranial Tumors with Special Reference to Its Occurrence in the Meningiomata, Surg., Gynec. & Obst. **55**:681, 1932.

3. Alpers, B. J., and Groff, R. A.: Parasellar Tumors: Meningeal Fibroblastomas Arising from the Sphenoid Ridge, Arch. Neurol. & Psychiat. **31**:713 (April) 1934.

4. Bailey, Percival: Intracranial Tumors, Springfield, Ill., Charles C. Thomas, Publisher, 1933.

practitioners and even ophthalmologists the condition is rare enough to merit attention. This fact warrants reemphasis of the cardinal features of the syndrome in the form of the following report of a case.

REPORT OF CASE

A youth, 17 years of age, of healthy appearance and with a normal personal and family history, was first examined on April 21, 1933, at the clinic for diseases of the eye of the New York Post-Graduate Medical School and Hospital. At that time he stated that he had been struck on the left side of the head six months previously and that soon afterward the left eye started to protrude and turn outward.

Vision of the right eye was corrected to normal with a minus sphere lens of 3.5 diopters. The visual field was normal; the fundus appeared normal, with the exception of a temporal conus. The left, or involved, eye had in the nasal portion of the field vision permitting the counting of fingers at 2 feet (60.96 cm.); the eyeball was turned outward and proptosed. The Hertel exophthalmometer

Fig. 1.—Picture of the patient on admission, showing the unilateral exophthalmos and deviation of the left eye.

registered exophthalmos of 10 mm. The motility of the left eyeball was partly limited in inward and upward rotation. No cephalic bruit was heard over the eye. Palpation of the eyeball and orbital ridge disclosed no abnormality. The tension was normal. The pupil was moderately dilated and reacted to light both directly and consensually. The sensibility of the cornea was normal. The olfactory sense was not involved. The third, fourth and sixth nerves were partially paralyzed. Examination of the fundus showed the disk to be grayish white, with its margin sharply outlined. A myopic crescent was present on its temporal margin. The retinal vessels appeared normal.

The patient was referred to the x-ray department for examination of the skull. Dr. Meyer's report was as follows: The skull showed no gross abnormality of the bones of the vault. The sella turcica was essentially normal. There was condensation of bone along the greater wing of the sphenoid on the left, with marked widening of the left sphenoid fissure. The picture was thought to be suggestive of a retrobulbar neoplasm with hyperostosis or condensation of the retro-orbital structures, notwithstanding widening of the sphenoid fissure.

The diagnosis was unilateral exophthalmos with simple optic atrophy and partial external ophthalmoplegia caused by a retrobulbar tumor arising within the orbital fossa.

Fig. 2.—Roentgenogram of the skull, showing the characteristic thickening of the retro-orbital plate on the side of the tumor. The left sphenoid fissure is greatly enlarged where the tumor has invaded it.

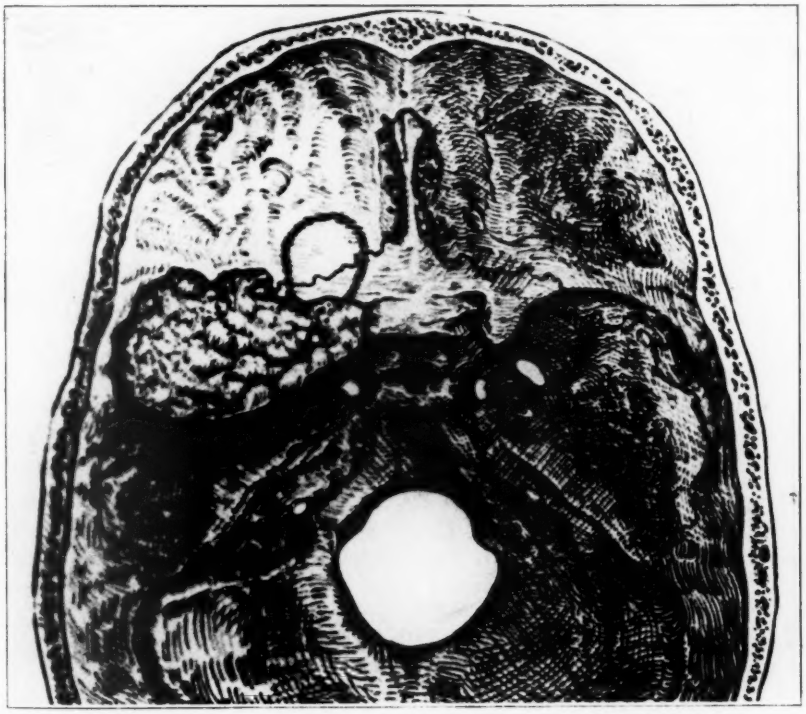

Fig. 3.—Drawing showing the approximate position and size of the tumor (after Bailey,[4] modified).

A neurologic consultation was obtained at this time from Dr. Scarff, who expressed the opinion that this case was one of intracranial tumor originating in the region of the sphenoid ridge. Dr. Meyer reported that the roentgen findings already described were compatible with this diagnosis.

On May 15 the patient was admitted to the service for diseases of the eye, where a complete medical examination was made. The history was unimportant except for some dizziness, occasional headaches and tinnitus at times in the left

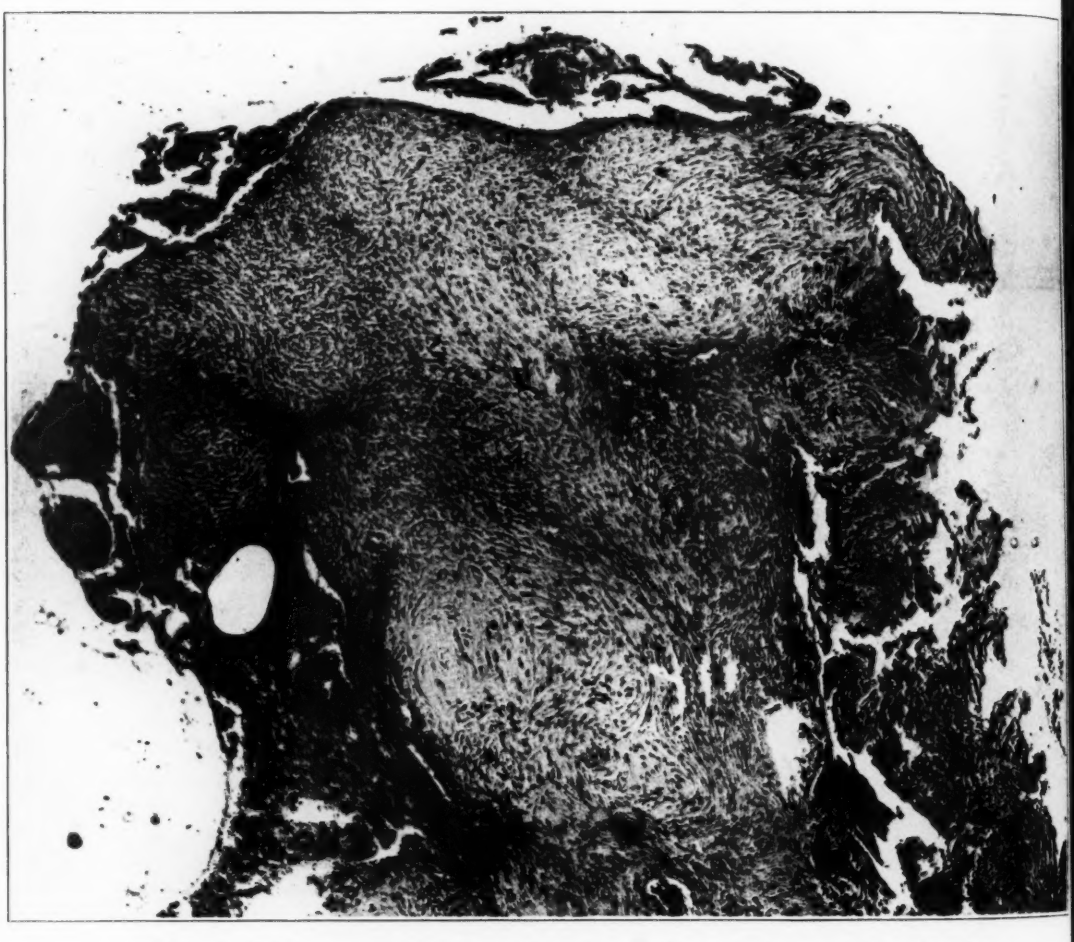

Fig. 4.—Low power photomicrograph of a section of the tumor showing its fibroblastic nature.

ear. The results of examination were entirely negative, except for the ophthalmologic and roentgen findings already mentioned. The laboratory reported that the basal metabolism, blood and urine were normal and that the Wassermann reaction of the blood was negative. As a result of the clinical observations and roentgen findings there still remained a difference between the ophthalmologic and neurologic diagnoses. It was decided to perform an orbital exploration for removal of the tumor or obtaining tissue for biopsy. It was thought, however, that if the orbital exploration was unsuccessful in exposing the tumor an intracranial operation could be performed later.

On May 16 an orbital exploration was performed by Dr. Cohen. This consisted in a long conjunctival incision in the lower outer culdesac, followed by blunt dissection, widening the opening and extending it to the orbital apex. The finger was then introduced into this opening with the object of feeling any abnormality, but none was found. The incision was closed with silk sutures, and at the end of six days the patient was discharged from the hospital without any improvement in the condition of the eye. It was considered at the time that a

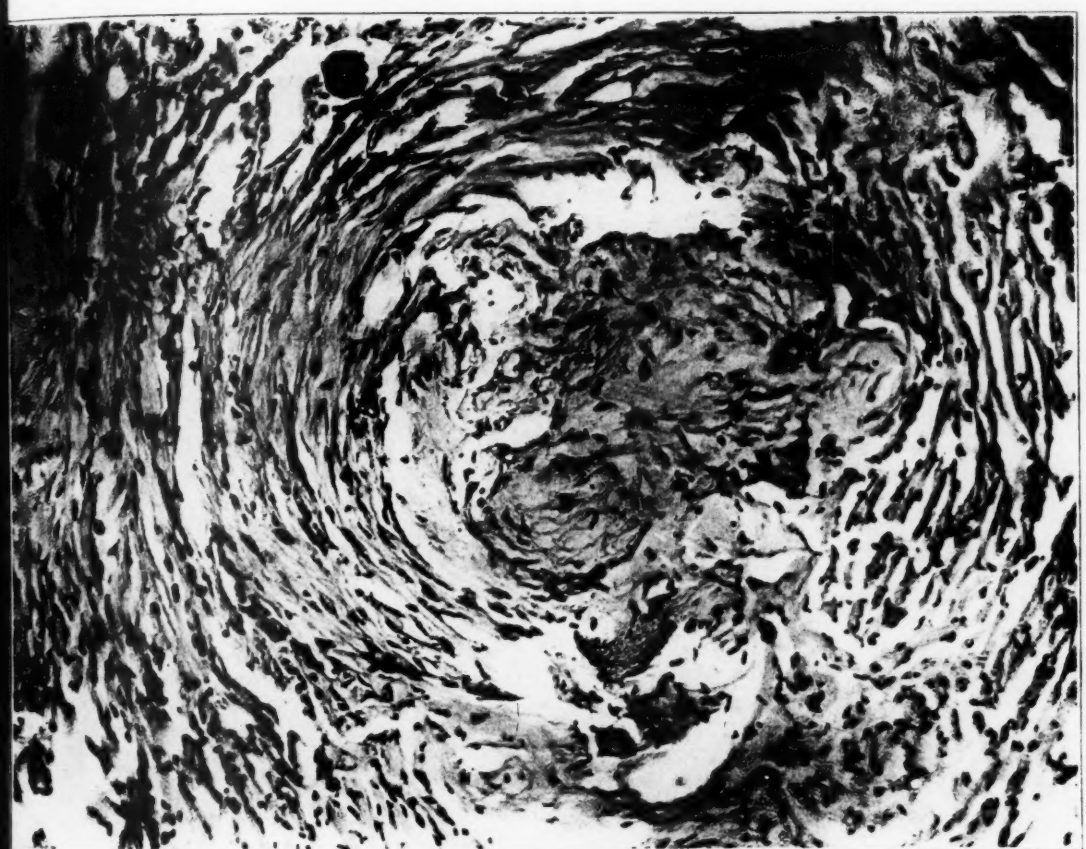

Fig. 5.—Section of the tumor under high power magnification, showing the tendency to whorl formation and the small deposit of calcium, or psammoma body.

flat orbital tumor might have been present in the orbit without being discovered by digital examination.

For a month the patient returned to the clinic for diseases of the eye for observation. The right eye remained normal. The left, or involved, eye showed a diminution in vision in the nasal portion of the field. Otherwise the condition remained unchanged.

The patient was referred again to Dr. Scarff, who made a second neurologic examination and came to the same conclusion as formerly, that the ocular condition was due to a meningioma of the left middle cranial fossa, situated close to

the sphenoid ridge. The patient was then readmitted to the New York Post-Graduate Medical School and Hospital in the neuro-surgical service, and on June 14 a cranial exploration was performed under procaine hydrochloride anesthesia (Dr. Scarff). A combined frontal and parietal bone flap was turned up without complication. The dura was widely opened; the temporal lobe was retracted upward as far as the middle fossa, and along the anterior wall of the fossa near the apex a flat, discrete, pebbly, grayish-pink tumor about 0.5 to 1 cm. thick was disclosed, It was flattened against the sphenoid plate and involved the sphenoid foramen. Apparently it had also invaded the dura and bone. It was from 0.5 to 1 cm. in thickness and from 4 to 5 cm. in circumference. As much of this mass as possible was removed piecemeal, but in view of the fact that the tumor had invaded the orbit through the sphenoid fissure, as was shown by roentgenograms, and as it had also infiltrated the bone of the base of the skull, complete extirpation was out of the question. Therefore only as much of the tumor as could be taken away without too great a risk was removed. A portion of the growth, roughly estimated at one sixth or one eighth of the total amount, was left close to the middle fossa and in immediate contact with the optic foramen, the sella turcica and the cavernous sinus. The patient stood the operation well and left the operating room in good condition.

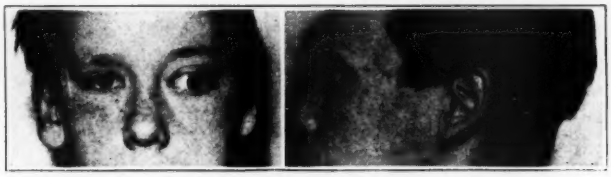

Fig. 6.—The patient as he appeared fifteen months after the operation. The eye looks essentially the same as before the operation.

A report by Dr. MacBrayer on the pathologic examination of the removed tissue was as follows: The mass of tissue was old, but young fibrous tissue showing areas of fibroblastic proliferation and appearing for the most part as whorls about blood vessels was also visible. In some fields three or four psammoma bodies were present, a few showing calcareous infiltration. Scattered here and there were clusters of endothelial cells, apparently normal and in general on the surface of the fibrous tissue. The latter probably originated in the dura mater, while the former came from the arachnoid. The striking feature was the large amount of fibroblastic tissue. The diagnosis was fibroblastic tumor of the meninges (so-called meningioma) presenting an unusually large amount of fibroblastic growth.

On Sept. 14, 1934, the patient was reexamined. He appeared to be in excellent health. The right eye had remained normal. The left eye showed a slight improvement as regards the exophthalmos; otherwise no ocular change was noted.

COMMENT

The term meningioma is applied by neurologic surgeons and neuropathologists to one of the more important types of intracranial tumor. Because it is almost invariably found attached to the dura mater it was originally thought to arise from this membrane and was first called

"dural endo-thelioma." Subsequent study has suggested that it may arise primarily from the arachnoid membrane, involving the dura only by secondary invasion. Because of this uncertainty as to the exact origin the general term meningioma has been employed more recently.

Pathologically, the tumor is essentially fibroblastic, resembling grossly the fibroid tumors found elsewhere in the body. It is firm and well encapsulated and never breaks through the capsule to invade the underlying brain tissue. Sooner or later, however, it traverses the dura and invades the contiguous bone. Even before any actual cellular invasion of the bone has taken place, however, periosteal irritation produced by the tumor causes hyperplastic overgrowth and thickening of the bone, which are highly characteristic of this type of tumor.

Microscopically, the tumor consists of elements which seem to be in part fibrous and in part endothelial. The cells tend to form into concentric whorls. It is not uncommon for calcium to be laid down in these whorls; hence the name psammoma, given by the early pathologists to this kind of tumor.

Clinically, intracranial meningiomas are characterized by certain features: (1) They occur essentially in adults; (2) they tend to arise from fixed points within the skull; (3) they produce definite and constant clinical syndromes, and (4) they have a low degree of malignancy. One of the points of election for intracranial meningiomas is the region of the sphenoid ridge and the anterior wall of the middle temporal fossa which forms a part of the posterior wall of the orbit. Here the meningioma assumes a flattened shape, often acquiring a thickness of only 0.5 to 1 cm., while spreading out in such a way as to cover a large surface of bone. For this reason the French authors have named this tumor meningioma *en plaque*. The tumor never invades the brain itself. Sooner or later, however, it invades the contiguous bone of the posterior orbital plate, producing the characteristic hyperplastic thickening already referred to, which, by encroaching on the orbital fossa causes slowly progressive proptosis of the eyeball, the most conspicuous feature of this syndrome. As the tumor extends medially along the sphenoid ridge it sooner or later reaches the optic foramen and by direct pressure on the optic nerve causes primary optic atrophy and blindness. The tumor also encroaches on the sphenoid fissure, eventually filling this fissure, enlarging it and making it irregular in shape, also paralyzing the nerves passing through it to the muscles of the eyeball and interfering with the return of venous blood from the orbit.

The diagnosis of meningioma of the sphenoid ridge rests on the following clinical points: 1. Slowly developing unilateral exophthalmos with external ophthalmoplegia and primary optic atrophy. 2. Roentgen evidence of hyperplastic thickening of the posterior orbital plate. The condition usually occurs in adult life.

The duration of the exophthalmos (which may be months or years) is of great importance in ruling out acute infections and vascular lesions in the large vessels communicating with the orbit.

The roentgen changes consist of hyperplastic thickening of the posterior orbital plate (the greater wing of the sphenoid) with widening of the sphenoid fissure. The first of these changes is essential in the diagnosis. They also tend to rule out a tumor within the orbital fossa, for when such a tumor produces any change in the bones of the orbit it is in the direction of pressure absorption with thinning of the bone. Only one other condition is likely to produce unilateral exophthalmos so slowly, namely, a fronto-orbital osteoma. But in this condition the roentgen rays make possible a clearcut differential diagnosis.

The prognosis must be qualified and must be considered from two standpoints: 1. Regarding life, the prognosis is favorable, for the rate of growth of the tumor is very slow, cases being known in which symptoms have been recognized for at least eight years before the condition had reached the point where the patient would consent to an operation. Owing to the unusually slow rate of growth, even should the tumor recur, it is not difficult to repeat the excision in later years. By this conservative method of treatment patients have been kept alive and free from serious intracranial symptoms for years and have eventually succumbed to disorders other than the intracranial disease. 2. Regarding eyesight, the prognosis is not so favorable, for the reason that the tumor has usually invaded the bony margins of the optic foramen and the sphenoid fissure by the time the patient sees the surgeon.

On the basis of the orbital signs and the roentgen findings, it is easy to mistake this condition for one originating within the orbital fossa itself. In our case a preliminary orbital operation was advised and performed, which failed to disclose the tumor. Whether a Krönlein operation on the orbit, by its greater exposure of the orbital contents, could have established a correct diagnosis it is impossible to state. This case shows how it is possible to mistake an intracranial tumor for one within the orbital fossa and is reported in the hope that our experiences may be useful to others meeting the same problem for the first time.

SUMMARY

Slowly progressing unilateral exophthalmos associated with ophthalmoplegia, primary optic atrophy, and hyperplastic thickening of the posterior orbital plate on the side of the exophthalmos, constitutes a definite clinical and pathologic syndrome produced by a meningioma (fibroblastoma) arising from the middle cranial fossa.

Various clinical and pathologic features of this syndrome are discussed.

A case illustrating the syndrome is reported.

HISTOLOGIC APPEARANCE OF RECENT RETINAL TEARS

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This communication deals with a case of idiopathic retinal detachment of short duration which became available for pathologic study. The number of cases in which eyes have been enucleated within three months after the onset of an idiopathic detachment is small, and because of the greater efficiency of the modern therapeutic methods the number is not likely to increase in the near future. The rarity of such pathologic reports may justify this paper.

REPORT OF A CASE

History.—A man, aged 54, of Anglo-Saxon ancestry, had spent most of his life in the Far East doing office work. He had had a moderate degree of myopia all his life and had worn lenses varying from —1.75 to —2.50 for each eye. About the middle of February 1934 he noticed small black spots in front of the right eye. On March 2 he became aware of a defect in the lower nasal quadrant of the visual field, accompanied by metamorphopsia. No injury or strenuous exercises of any kind had preceded the onset of the ocular symptoms. The patient was admitted to the Peiping Union Medical College Hospital on March 14. The general medical history and findings were insignificant except for osteomyelitis of the right tibia, which had healed at the age of 5, leaving a slight deformity of the right leg.

Examination.—On the day of the patient's admission to the hospital the detachment had the appearance of a distended balloon which occupied almost the entire upper half of the right retina. The lower border of the detachment was arc-shaped, starting on the nasal side in the 45 degree radius, covering the disk and ending on the temporal side in the 160 degree meridian. The most prominent part of the retina had a refraction of plus 16 diopters. The detached retina was dark gray. Its surface was smooth, except for a few white ridges which ran irregularly across its surface. Several large retinal tears were visible. Starting on the temporal side one could see an approximately elliptic tear extending from the 130 to the 160 degree radius. At first glance this tear looked like a detachment, because of its very peripheral location and its smooth arc-shaped posterior border. More careful examination under extreme dilation of the pupil revealed that the anterior edge of the tear was visible with the ophthalmoscope and that anterior to that edge two or three small oblong tears, the longer diameter of which lay in the meridional direction, were also present. Going along the equator toward the nasal side, two more large, approximately round tears, with ragged edges, became visible. They were located near the 125 degree radius, in the neighborhood of the equator. Near the nasal boundary of the detachment in the extreme periphery another small round tear could be made out.

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The detachment responded promptly to rest in bed. After two days the retinal balloon collapsed. The disk, the macula and the true borderline of the detachment became visible. Some fine lesions of the type of drusen occupied the macular area. The lower edge of the detachment now lay from 20 to 25 degrees above the horizontal meridian. The most prominent point of the detachment had a refraction of plus 8 diopters. Most of the detached portion of the retina had become translucent. The tears were still clearly visible but had changed their location with relation to the other structures of the eye. They were all found to be located within 3 disk diameters from the peripheral border of the ophthalmoscopic field.

The rest of the remaining fundus was thought to be normal, except for what looked like an old choroiditic lesion near 6 o'clock, in the extreme periphery. The intra-ocular tension was only 12 mm. of mercury in each eye.

The multiplicity of the tears suggested a delicate and friable condition of the retina.

Operation and Course.—On March 19, 1934, the right eye was operated on. The method of Šafář was employed, and his two and three point electrodes were used. One double barrier of coagulated areas was laid along the equator of the upper temporal quadrant, beginning in the 90 degree radius and ending at the upper border of the external rectus muscle. At those two places as well as in the 125 degree radius the barrier was connected with the ora serrata. In addition to this, a shorter barrier was laid over the upper half of the nasal quadrant. A large amount of subretinal fluid escaped on removal of the electrodes. Beside this, two oblique posterior sclerotomies were made with the Graefe knife anterior to the barrier. Only a small amount of fluid escaped through these holes. The superior rectus muscle, the insertion of which had been severed at the beginning of the operation, was sutured back in place. At the end of the operation it was noticed that the retina had flattened considerably, but it had not become completely reattached.

Two weeks after the operation, a narrow zone of detachment was still present immediatley posterior to the area of the operation. The latter presented the usual appearance: In the beginning there were round areas of retinal edema, which slowly cleared up and revealed the choroiditic lesions. The remaining detachment confined itself to a more or less horizontal band about 3 disk diameters in height, which followed the barrier fairly closely. The tears were no longer visible; at least, no actual retinal defect could be discerned between the multiple choroiditic lesions, nor could any new tear be discovered elsewhere in the fundus. The patient's vision, with a —4 lens, was 6/20, and he could read Jaeger's test type no. 6. He still complained of pronounced metamorphopsia. The visual field showed a defect in the lower portion which corresponded with the area of the operation and the adjoining detachment. No change in the picture of the fundus or subjective findings occurred for another week, so that the patient was temporarily discharged from the hospital. A few hours before he left it was found that the defect in the visual field extended, in the shape of a tongue, into the upper nasal quadrant. No corresponding change in the extent of the detachment could be seen with the ophthalmoscope. The patient was instructed to watch his field very carefully, and, according to his statements, which I have every reason to accept as accurate, the condition of the right eye remained unchanged or improved slightly until June 3, when he slipped and, although he did not actually fall, gave himself a rather violent jerk. A few minutes afterward he noticed a new visual defect in the upper portion of his right field. He returned to the hospital on June 6. A new retinal detachment occupying most of the lower half of the fundus was present, while the

upper half was in place up to the border of the area of the operation. I thought that the localized detachment above, which had been present when the patient was discharged from the hospital, had moved downward. More careful examination, however, led to the discovery of what looked like three new tears. Because of increased cloudiness of the vitreous, the fundus was not as clearly seen as on previous occasions. Two of the tears were spindle-shaped and were located below and slightly nasally to the disk in an area which had previously shown no visible pathologic change. The third tear seemed to be located near 6 o'clock in the area of the old choroiditic lesion which I had seen before. Now, however, I thought the retina had become detached over the entire extent of that choroiditic lesion, because there seemed to be an intensely white arc-shaped borderline which formed the posterior border of an area considerably lighter than the rest of the fundus. During the patient's few days' stay in the hospital, the defect of the visual field rapidly became more pronounced and the extent of the detachment increased. The new tears seemed to me to forecast further unfavorable developments. The patient, a very high-strung and nervous person, could not face another operation with such meager prospects of success; moreover, another period of three or four weeks in the hospital would have hurt his career considerably. Therefore he asked to have the eye removed. This was done on June 10.

The eyeball was fixed in Zenker's fluid and embedded in pyroxylin (celloidin). A small nasal calotte was removed, and vertical serial sections, 17 microns thick, were made.

Comment on Clinical Findings.—The large number of the tears present in this case four weeks after the onset of the ocular symptoms, their size and their ragged edges clearly indicated that the patient's right retina had been badly and extensively damaged by previous inflammatory disease, and perhaps other diseases. Why the retina did not become reattached after the operation was not clear to me. I felt that the barrier was complete. The area anterior to it was transformed into a diffuse chorioretinal scar which allowed no definite statement as to the presence or absence of the retina in any particular portion. Considering the prompt collapse of the balloon after rest in bed, it seemed reasonably certain to me that no pulling action of the vitreous prevented the complete reattachment in that part of the fundus. The most probable cause of the persistent partial detachment was, therefore, either shortness of the upper part of the retina resulting from the extensive defects plus postoperative loss of tissue or faulty operative adhesions between noncorresponding retinal and choroidal areas. The detachment below, which developed acutely three months after the onset of the original detachment, could be explained either as caused by the usual downward spreading of a detachment in the upper half of the globe or as a new detachment (*récidive*, according to Gonin [1]). The picture in the visual field at the time of the discharge of the patient after the first operation and common experience spoke in favor of the first assumption, while

1. Gonin, J.: Les rechutes et récidives du décollement rétenien, Arch. d'opht. **48**:487 (July) 1931.

the development of tears in areas which had been apparently normal on previous ophthalmoscopic examinations strongly supported the second possibility. Whatever the cause of the detachment below, one fact was definitely established by the clinical examination of the patient, viz., that tears had developed in an area which eight weeks before had been normal on ophthalmoscopic examination. The patient's own observation made it at least highly probable that the tears and the detachment below had developed only a few days before the eye was removed. Thus, the histologic observations made in this case pertain to one of the earliest stages of a tear ever studied pathologically.

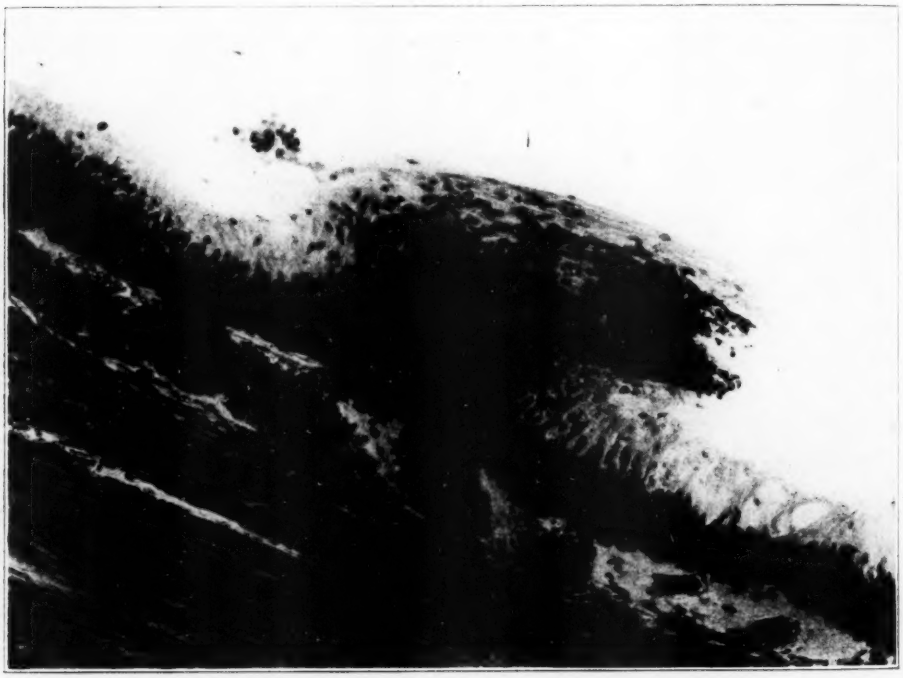

Fig. 1.—Photomicrograph showing an excrescence formed by the epithelium of the pars plana near the field of operation. Magnification, × 155.

Pathologic Observations.—Anterior Segment: The cornea, iris, anterior chamber and iridocorneal angle were free from pathologic changes. The lens was normal.

The anterior chamber was deep, and the ciliary muscle was of the myopic type. Except for two small groups of lymphocytes near the base of two ciliary processes, there were no signs of active or inactive iridocyclitis. The ciliary epithelium, the pigmented as well as the nonpigmented, mainly of the pars plana, had proliferated somewhat toward the interior of the eye and also into the substance of the ciliary body. Thus at two places of the pars plana mushroom-like excrescences had formed (fig. 1). One lay in the neighborhood of the choroiditic lesion at 6 o'clock, the area referred to in the description of the fundus; the other adjoined the field of the operation. Outside of these two places, the proliferation was only

slight, of the type described by Kerschbaumer [2] and Kuhnt,[3] and hardly comparable to the extensive hyperplasia described by Leber [4] in eyes presenting retinal detachments.

Area of the Operation: In the episcleral tissue the inflammatory process had almost subsided. The sclera was still necrotic over a large area. The process of repair was clearly visible in the form of young granulation tissue which extended from healthy unaffected portions of the sclera into the necrotic areas. The places where the pin electrodes had penetrated the sclera (*Stichkanaele*) were still plainly visible and were filled with granulation tissue which took its origin from the episcleral tissue. All these "channels" seemed to be completely closed, allowing no leakage of fluid. Granulation tissue also lined and invaded the necrotic sclera from the inside. Next to it, and seemingly inseparable from it, lay the choroidal scar.

The scar consisted essentially of layers which paralleled the lamellae of the sclera. The outer layers were made up of young connective tissue containing few chromatophores, some free pigment and a small number of infiltrative cells. A considerable number of small hemorrhages were embedded in these layers. In the inner portion clumps of free pigment prevailed. Only small fragments of Bruch's membrane could be discerned. Remnants or derivatives of the pigment epithelium could not be recognized with certainty. There were, however, places in which epithelial (?), nonpigmented cells had proliferated on the inner surface of the choroidal scar.

The appearance of the retina varied a great deal. Over most of the equatorial portion of the upper temporal quadrant the choroidal scar was covered by only one or two irregular layers of glia cells. Toward the 90 degree radius, however, retinal structure became visible. There were areas within the immediate field of the operation where the choroid was completely transformed into pigmented scar tissue, where the only remnants of the pigment epithelium were clumps of loose pigment but where the structural elements of the retina were apparently well preserved. Only the rods and cones were visibly damaged. They were irregular in shape, and amorphous, eosinophilic masses lay between them. In other places it could be seen that the retina had actually become included in the choroidal scar. There again it was amazing to see that in the immediate neighborhood of such a complete fusion between the retina and the choroid the adjoining retina was well preserved (figs. 2 and 3).

Several retinal tears and old atrophic areas in the retina could be identified as such in the field of the operation. They are not described here in detail because they do not present the true picture either of changes present in retinal detachment before operation or of operative changes.

Remainder of the Eye: The remnants of the vitreous occupied the space between the lens and a frontal plane laid through points midway between the ora serrata and the equator. The appearance of the vitreous was, as far as I could see, the one usually seen in eyes which have been fixed in Zenker's fluid. The only unusual feature was the presence of edematous pigmented epithelial cells, about 20 microns in diameter, between the fibrils of the vitreous precipitate. The pigment content of these cells varied greatly. In some of them only brown granules but no nuclei were visible. These cells had a tendency to form clusters.

The choroid was moderately hyperemic throughout. A ring of lymphocytic infiltration of the choroid surrounded the field of the operation. Smaller fresh

2. Kerschbaumer, R.: Arch. f. Ophth. **34**:16 (Dec.) 1888.

3. Kuhnt, H.: Ber. d. deutsch. ophth. Gesellsch. **13**:38, 1881.

4. Leber, T., in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1916, vol. 7, p. 1548.

Fig. 2.—Photomicrograph showing the edge of a tear within the field of operation. The choroid is transformed into a pigmented scar. There is no definite proliferation of the pigment epithelium. One *Stichkanal* goes through the center of the picture. There is partial necrosis of the sclera around it. Repair was made from the lower left hand croner. Magnification, × 44.

Fig. 3.—Photomicrograph showing the border of the field of operation. A *Stichkanal* goes through (in this section) the outer half of the sclera. The sclera that lies to the left of this canal is still completely necrotic. Of the right half, approximately the inner two thirds are replaced by young granulation tissue, while the outer third is necrotic. The left half of the choroid and retina is transformed into a pigmented scar with hemorrhages and a moderate amount of infiltrative cells. The right half of the retina and choroid is, despite its location near a *Stichkanal*, fairly well preserved. Magnification, × 44.

infiltrates, also consisting of small lymphocytes, were located at several other places. The retina was completely detached; except for one large convolution below the macula and the disk, the inner surface of the detachment was fairly flat. The outer layers, however, formed innumerable mushroom-like elevations into the subretinal space which were most pronounced around the posterior pole and gradually disappeared in the periphery. These elevations seemed to be caused by edema, for the inner nuclear and the outer plexiform layers contained many empty spaces which in vivo were filled with a fluid poor in protein. The greater degree of edema in the outer layers of the retina created the impression of a discrepancy in length between the inner and the outer surface of the retina.

No foreign cells or membranes could be discerned anywhere on the inner retinal surface. Only at one place, in the lower nasal quadrant, a group of about 20 glia cells was lying on the retina, loosely attached to the inner limiting membrane.

Near 6 o'clock I expected to find an old choroiditic lesion and a retinal detachment. Only the presence of the former was confirmed. Near the vertical meridian, between the ora serrata and the equator, the choroid showed diffuse but not severe atrophy. The pigment epithelium was irregular, almost void of pigment at some places and proliferating at others. The choriocapillaris was nearly obliterated, while the remaining vascular layers were only slightly atrophic, containing one fresh infiltrate. The adjoining ora serrata was atypical: on the one hand because of proliferation of ciliary epithelium and on the other because of glial extensions of the retina which overlapped a portion of the pars plana (fig. 4). The retina over the whole preequatorial region from 6 to 7 o'clock was devoid of visual epithelium. The nuclear layers were well preserved. Blessig's grooves were only moderately developed and were limited to the immediate neighborhood of the ora serrata. A definite retinal lesion was located slightly anterior to the equator, approximately corresponding to the posterior border of the old choroiditic lesion. This retinal lesion had the shape of a narrow band the cross-sections of which could be followed through sixty vertical sections. Its nasal and temporal ends lay considerably farther forward than its middle portion. The lesion was essentially atrophic, containing only a few cells. The retina was replaced by a dense meshwork of glia fibers in which some pigment had become deposited. In some places the lesion had a looser texture with small vacuoles; in others, owing to the absence of the inner layers, the retina became very thin. In four sections the entire thickness of the retina was taken up by one empty cystic space the walls of which seemed to be formed by a single or double layer of glia cells or fibers (fig. 6). A complete rent of the retina was not present in that region.

The vitreous did not seem to be connected with the retina over the lesion.

The detachment which I had diagnosed with the ophthalmoscope proved to be a lesion which might easily have developed into a complete rent but had actually not done so.

The two tears, below and nasally to the disk, which had been seen in vivo, were verified by microscopic examination. Both were spindle-shaped and lay radially to the disk. The choroid behind them was normal. The surrounding retina was carefully searched for pathologic changes but aside from the aforementioned edema and a slight thinning of the inner layers, nothing was found. In the vertical sections the tears appeared as narrow clefts, the inner apertures of which were filled by amorphous eosinophilic masses. Under the microscope these masses looked much like the coagulated subretinal fluid. The edges of the tears were rolled inward to such an extent that in some places rods and cones were actually lining the lateral walls of the clefts, including the marginal portion

Fig. 4.—Photomicrograph of the retina at the ora serrata which has formed a process consisting mainly of glia fibers which bridge over a portion of the pars plana and then become fused with an excrescence of the ciliary epithelium. Magnification, × 119.

Fig. 5.—Photomicrograph showing one cystic space occupying the entire thickness of the retina (taken from the region of the atrophic retinochoroiditic lesion near the equator at 6 o'clock). Note the clumps of pigment (apparently not intracellular) lying on the inner surface of the retina. Magnification, × 155.

Fig. 6.—A cross-section through the two spindle-shaped rents. Note the pronounced edema in the outer retinal layers. Magnification, × 44.

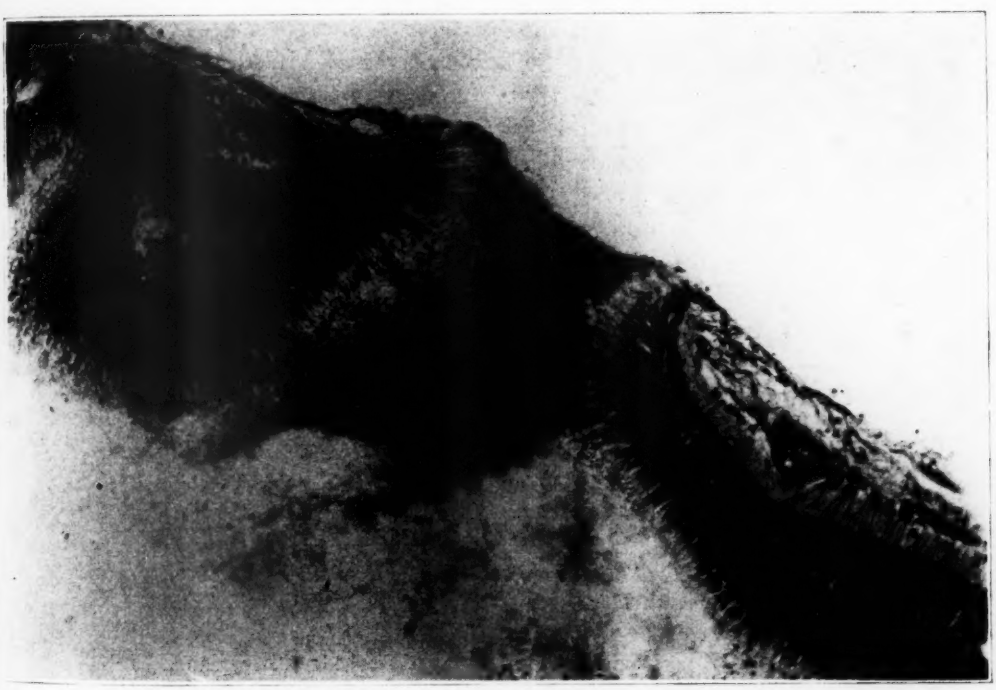

Fig. 7.—A cross-section through one of the spindle-shaped tears. The inner aperture is filled with an amorphous mass which stained heavily with eosin. At the edges of the tears the retina is ectropionized. There visual epithelium lies on the anterior surface of the retina. The continuity between the membrana limitans externa and the membrana limitans interna is visible. Magnification, × 119.

of the anterior surface of the retina. In other places the visual epithelium had come off, and the cleft was lined by the external nuclear layer. In both cases the external limiting membrane could be followed from its normal position, anterior to the visual epithelium, along the lateral wall of the clefts onto the anterior surface of the retina where it seemed to fuse with the membrana limitans interna. This fusion was, at some places, so complete that it gave the impression of one continuous membrane. Around the tears right under the membrana limitans interna lay glia cells in unusual numbers. The remainder of the retina was normal, except for a few small slightly atrophic areas.

Comment on Pathologic Observations.—So far as these observations pertain to the field of the operation, they only supplement the reports made by Šafář,[5] Fischer[6] and Stallard.[7] It was interesting to see that the necrosis of the sclera was only partly repaired two and a half months after operation. The fact that the retinal structure was so well preserved in a number of places corroborated Šafář's ideas about the relative harmlessness of the short pin electrodes. The particular retinal area destroyed by the insertion of the electrode and by the consecutive retinochoroidal adhesion was small, and the surrounding retina retained its gross histologic structure fairly well.

Aside from this, the pathologic changes in my case can be compared with those obtained in the few other cases of retinal detachment (Nordenson,[8] Kümmell,[9] Redslob[10] and Sourdille[11]) which likewise became available for anatomic studies shortly after the onset of the detachment. Here I can hardly add anything to the observations of the aforementioned authors. Like some of them, I found the sequelae of inflammatory or senile-degenerative processes in the retina and choroid which weakened the retinal structure so that tears had, or could easily be, formed. The actual extent of such changes in my case was, of course, much greater than that described here because most of the

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5. Šafář, K.: Behandlung der Netzhautabhebung mit multipler diathermischer Stichelung. Abhandlungen aus der Augenheilkunde und ihren Grenzgebieten, Berlin, S. Karger, 1933, p. 16.

6. Fischer, F.: Anatomische Befunde zur Behandlung der Netzhautabhebung mit multipler diathermischer Stichelung, Ztschr. f. Augenh. **83**:95 (April) 1934.

7. Stallard, H. B.: Histologic Appearance of an Eye Successfully Treated by Diathermy for Retinal Detachment: Fatal Termination from Pulmonary Thrombosis on Nineteenth Day After Operation, Brit. J. Ophth. **17**:294 (May) 1933.

8. Nordenson, E.: Die Netzhautablösung, Wiesbaden, Bergmann, 1887.

9. Kümmell, R.: (*a*) Zur Entwicklung der Netzhautabhebung, Arch. f. Augenh. **95**:214, 1925; (*b*) Zur Anatomie der frischen Netzhautablösung, ibid. **100-101**: 314, 1929.

10. Redslob, E.: Contribution à l'étude de l'anaotmie pathologique et de la pathogénèse du décollement rétinien, Bull. Soc. d'opht. de Paris, 1931, p. 211.

11. Sourdille, G.: Congrès Soc. franç. d'opht., Paris **45**:236, 1932.

previous pathologic changes had been in the area of operation where they could not be clearly differentiated from operative effects on the tissues. I found no evidence in favor of Leber's theory of preretinitis. The remnants of the vitreous were not found to be adherent to the retina. What rôle the vitreous played in the mechanism of the detachment could not be detected by microscopic studies of the fixed specimen, and must be left to conjecture on the basis of the various experimental and clinical findings now available.

Not much weight can be attached to the few fresh choroiditic infiltrations which I found at various places. Infiltrations of this type and size are too common in eyes without retinal detachment to serve as a clue in the solution of the problem of detachment.

My observations show clearly how a retinal tear can develop from an atrophic retinal lesion which is the result of retinochoroiditis. My sections, however, do not show clearly how the two spindle-shaped tears developed. The patient was such a keen observer that I have good reason to believe that those tears were recent, probably only of a few days' duration. There seemed to be only vague signs of previous pathologic changes in that region. The inward-rolling edges of the tear could be explained in many different ways, the most obvious being the pronounced edema of the outer layers of the retina which made them relatively longer than the corresponding inner layers. The only detail which cannot be explained on this basis was the apparent continuity of the external and internal limiting membranes of the retina. Schematic considerations of the arrangement of the retinal layers on the basis of the "edema theory" lead me to the conclusion that the distance between the edge of the tear in the internal limiting membrane and that in the outer nuclear layer represents the thickness of the retina at the place where the tear formed, irrespective of the position of the retina. If that distance is as short as it is in my specimen, namely, almost nil, then it is certain that the retina was very thin in that region before the formation of the tear. In other words, there were previous pathologic changes, mainly in the inner layers. Those changes were either invisible to the ordinary ophthalmoscope, or they developed during the eight weeks between the first discharge of the patient and the second admission.

With regard to the probable cause of these changes, it can only be stated that they were not due to a primary choroiditis. The presence of two tears and the short duration of the detachment in the lower half spoke against distention by a subretinal transudate. What rôle the other possible mechanical factors like pulling action on the part of the vitreous, the ocular movements, a shrinking process within the retina itself or a

disturbed hydrostatic pressure equilibrium (Kümmell[9b] and Baurmann[12]) may have played as primary or precipitating causes, I am, like Arruga,[13] not able to tell. The part of the vitreous, or a shrinking process within the retina itself, seems more probable.

Of the other recent tears reported, only that in Kümmell's second case (enucleation seven weeks after the onset of the detachment[9a]) resembled mine. In the other cases the neighboring retina was too degenerated or the description too brief to allow a comparison.

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12. Baurmann, M.: Ueber Netzhautablösung, zugleich ein weiterer Beitrag zum Studium der Eigenschaften des Glaskörpers, Arch. f. Ophth. **122**:415 (July) 1929.

13. Arruga, H.: Ueber die Entstehung der Netzhautrisse und der idiopathischen Netzhautablösung, Klin. Monatsbl. f. Augenh. **93**:43 (July) 1934.

THE CENTRAL PATH OF THE LIGHT REFLEX

A STUDY OF THE EFFECT OF LESIONS

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The constriction of the pupils in response to light is a reflex that has long been of interest to physicians because its loss is a cardinal feature of the well known Argyll Robertson syndrome. Although an extensive literature has accumulated in the past forty years on the Argyll Robertson phenomenon, its mechanism still remains one of the unsolved problems in neurology. It is apparent that a major obstacle to an explanation of the syndrome has been the uncertainty regarding the localization in the brain stem of the pathway of the light reflex.

A dorsal view of the brain stem (fig. 7) shows some of the structures which must be taken into consideration in any discussion of this pathway. The superior and inferior colliculi of the corpora quadrigemina are situated dorsal to the cerebral aqueduct and form the roof or tectum of the midbrain. Between the superior colliculus and the thalamus there is a transition zone known as the pretectal region, which in the brain from which the photograph was made had been destroyed by electrolysis. The lateral geniculate body receives fibers from the optic tract, but many of the optic fibers are continued past this nucleus into the brachium of the superior colliculus, which reaches the lateral border of the rostral part of this eminence. We shall present evidence that some of the fibers of the brachium are continued rostrally past the superior colliculus into the pretectal region.

Certain features of the course taken by the light reflex are already known. The work of Karplus and Kreidl [1] on the cat and monkey established the fact that the constrictor pathway runs centrad from the optic tract in the brachium of the superior colliculus. In spite of the large amount of contrary evidence, the erroneous view has long been held that the constrictor impulses pass from the brachium into the substance of the superior colliculus, and thence by way of the tectobulbar fibers to the sphincter portion of the oculomotor nucleus. Maintenance of the light reactions after extirpation of the superior colliculus, as verified by a series of workers, opposed this view, however, and with the tectum thus excluded, the question remained as to the path of the

From the Institute of Neurology, Northwestern University Medical School.

1. Karplus, J. P., and Kreidl, A.: Ueber die Bahn des Pupillarreflexes (Die reflektorische Pupillenstarre), Arch. f. d. ges. Physiol. **149**:115, 1912.

constrictor impulses from the brachium of the superior colliculus to the sphincter nucleus of the oculomotor.

The answer to this question was given in a recent paper (Ranson and Magoun [2]) which reported the results obtained from a systematic stimulation of the interior of the caudal part of the diencephalon and the rostral half of the midbrain in normal casts. This stimulation was carried out with the aid of a refined technic employing the Horsley-Clarke stereotaxic instrument. For the details of this investigation, the original paper should be consulted, but a summary of the path of the pupillary light reflex, as traced by this method, is illustrated diagrammatically in figure 1. The light reflex pathway is seen to run

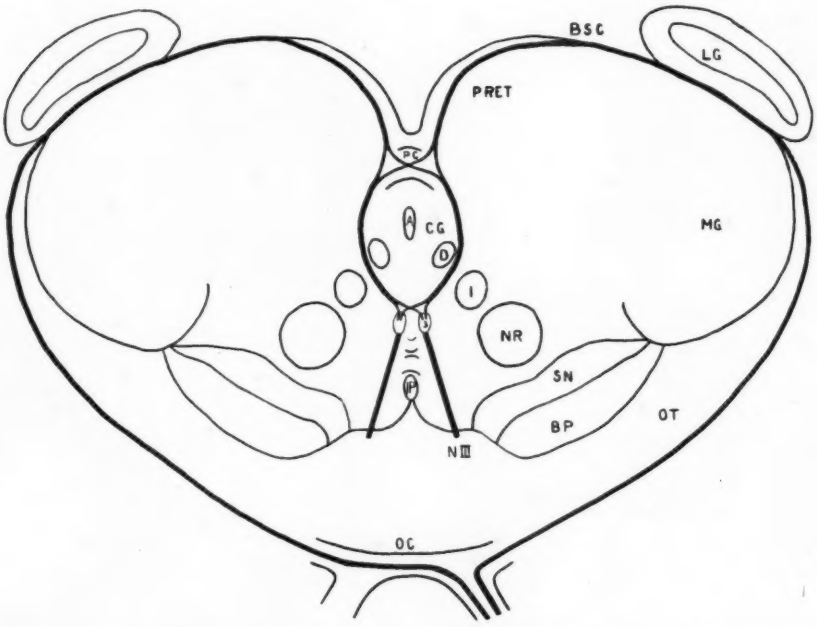

Fig. 1.—Diagram of the path of the pupillary light reflex. The location of synapses is not shown. The abbreviations used in figures 1 to 7 are as follows: *A*, aqueduct; *BP*, basis pedunculi; *BSC*, brachium of superior colliculus; *CG*, central gray matter; *D*, nucleus of Darkschevich; *H*, habenula; *HP*, habenulopeduncular tract; *I*, interstitial nucleus; *IC*, inferior colliculus; *IP*, interpeduncular nucleus; *LAT VENT*, lateral ventricle; *LG*, lateral geniculate body; *MG*, medial geniculate body; *NR*, red nucleus; *N III*, oculomotor nerve; *OC*, optic chiasma; *OT*, optic tract; *PC*, posterior commissure; *PP*, pulvinar pars posterior; *PRET*, pretectal region; *SC*, superior colliculus; *SN*, substantia nigra; *THAL*, thalamus; *VEN III*, third ventricle; *VEN IV*, fourth ventricle; *XSC*, commissure of the superior colliculus; *3*, oculomotor nucleus.

2. Ranson, S. W., and Magoun, H. W.: The Central Path of the Pupilloconstrictor Reflex in Response to Light, Arch. Neurol. & Psychiat. **30**:1193 (Dec.) 1933.

centrally in the optic tract on the lateral surface of the brain stem and ventral to the lateral geniculate body into the brachium of the superior colliculus. From the brachium the pathway runs medially into the pretectal region, and thence with a partial crossing both in the posterior commissure and ventral to the cerebral aqueduct, it swings ventrally around the rostral central gray matter to the oculomotor nucleus.

Behr,[3] in his monograph on pupillary activity, presented a reconstruction of the light reflex pathway in which the location of its course from the brachium of the superior colliculus to the oculomotor nucleus, together with the site of its central decussation, were not indicated, since at that time, as Behr stated, this course was only conjectural. The diagram presented in figure 1 completes the knowledge of the path of the light reflex, which is seen to run centralward into the pretectal region, rather than into the superior colliculus, and to reach the oculomotor nucleus after a partial central decussation both in the posterior commissure and ventral to the central gray matter of the cerebral aqueduct.

If this diagram correctly represents the path of the pupillary light reflex, it should be possible to interrupt it by making lesions in the pretectal region. Lesions in this situation have been made in cats with the aid of the Horsley-Clarke apparatus, and the results obtained are reported in the present paper.

METHODS

The observations to be reported were made on cats with pretectal lesions produced with the aid of the Horlsey-Clarke stereotaxic instrument. This instrument makes it possible to place the bare tip of an otherwise insulated electrode at any desired point in the interior of the brain, and to destroy the structures located at that point by electrolysis. With modifications in technic the same instrument may be utilized for stimulating the interior of the brain. The use of the stereotaxic instrument has been described elsewhere (Ranson[4]).

Before a lesion was made in the brain, the cats were placed under deep anesthesia with pentobarbital sodium (40 mg. per kilogram of body weight). Under aseptic conditions about one-half square inch of bone was removed from the parietal region of the skull, and the stereotaxic instrument was adjusted to the animal's head.

The attempt was made in all cases to produce a lesion of the pretectal region without damage to the superior colliculus lying immediately caudad. Lesions of the caudal part of the thalamus involving only the most rostral portion of the pretectal region were produced in eighteen cats, which were kept for from three to five weeks. Bilateral pretectal lesions were made in eight cats, which were kept for from three to twelve weeks. Unilateral pretectal lesions were made in eight cats, which were kept for from one to nine weeks.

3. Behr, C.: Die Lehre von den Pupillenbewegungen, Berlin, Julius Springer, 1924.

4. Ranson, S. W.: On the Use of the Horsley-Clarke Stereotaxic Instrument, Psychiat. en neurol. bl. (Feestbund. C. U. Ariëns Kappers) **38**:270 (May-Aug.) 1934.

At the close of the operation the Horsley-Clarke instrument was removed from the head, the wounds were closed, and the nictitating membranes were removed from both eyes in order to facilitate subsequent examination of the pupils. The light reactions were usually tested every day for the first week and two or three times a week subsequently until the time of sacrifice.

The light reactions were examined with the animal resting comfortably in a hammock in a dimly lighted room. The eyelids were held open to expose the pupil and the width of the pupil was measured in millimeters before and during lighting, for both the direct and the consensual reactions. With the degree of illumination used, the pupils of a normal cat would constrict from their previous width to a slit measuring 1 mm. or less in width. A reaction of this amount may therefore be referred to as the normal light reflex.

In the case of some of the cats two additional operative procedures were performed. Four of the cats with bilateral lesions and one of the cats with a unilateral lesion were subjected to bilateral cervical sympathectomy under aseptic precautions from four to twelve weeks after the initial pretectal lesions had been made. The seventh nerves of these and two other animals were bilaterally sectioned, without aseptic precautions, on the day before the animal was put to death.

After the animal was put to death, the extent of the injury to the brain was determined microscopically and was plotted in each case on a standard series of six transverse levels from the diencephalon, pretectal region and superior colliculus of a normal cat's brain and was represented also on a view of the dorsal surface of the brain stem. In addition a written description of the lesion and a tabulation of the light reactions following operation were made for each experimental animal. In this way thirty-four condensed protocols, each consisting of a tabulation of the light reactions following operation and a written and graphic description of the lesions, were obtained.

OBSERVATIONS CONCERNING THE EFFECT OF THALAMIC AND PRETECTAL LESIONS ON THE LIGHT REFLEX

This study may be separated into three series of chronic experiments dealing with the effect on the light reflex (1) of lesions of the caudal part of the thalamus, (2) of bilateral lesions of the pretectal region and (3) of unilateral pretectal lesions. In addition, information of a general character has accumulated from all three of the series, but chiefly from the second, bearing on the reflex constriction of the pupil from causes other than increased intensity of light, on the dilatation of the pupils effected by an inhibition of the constrictor portion of the oculomotor nucleus, and on the effect of chronic irritation of the light reflex path on the width of the pupils. As a large part of this study has had to do with amounts of impairment in the light reaction, the method used for making quantitative estimates of such impairment should first be discussed.

Determination of the Light Reactions.—As has been stated under the heading "Methods," the light reactions were tested by measuring the width of the pupils in millimeters before and during lighting with a fountain pen flashlight. The degree of illumination produced was sufficiently intense to cause a constriction of the pupils of a normal cat to a width of 1 mm. or less. In the dimly lighted room in which these

tests were made, the pupils before lighting were fairly dilated, measuring on the average about 9 mm. in width. On lighting the eye of a normal cat, the pupil would constrict from 9 mm. to 1 mm. or less in width, giving a change of 8 mm. On lighting the eye of an experimental cat—for example, one in which a small bilateral pretectal lesion had been made—the pupil would constrict from a width of 9 mm. to a width of only 5 mm., giving a change of 4 mm. This excursion of 4 mm. represents only one half of the normal excursion of 8 mm., and the light reaction of this cat may, therefore, be said to be reduced to one half of the normal light reflex.

In the descriptions of impairment of the light reactions in the following observations the terms used have the following significance:

A trace of reaction equals one eighth of the normal.
A slight reaction equals one fourth of the normal.
A fair reaction equals one half of the normal.
A good reaction equals three fourths of the normal.
A normal reaction equals constriction to 1 mm. or less.

TABLE 1.— *Cat 22. Operated on September 21*

Date	Width of Pupil Before Lighting		Lighting Right Eye		Lighting Left Eye	
	Right	Left	Direct	Consensual	Direct	Consensual
September 23	1.5	2	No reaction		No reaction	
September 26	3	4	Trace	Trace	Trace	Trace
October 5	4	4	Fair	Fair	Fair	Fair
October 6	8	9	Normal		Normal	

The experiments dealing with the effect on the light reflex of lesions in the caudal part of the thalamus may now be described.

Lesions of the Thalamus.—Since in all of the cats with pretectal lesions the mediocaudal portion of the thalamus was also materially damaged, it is important to rule out thalamic injury as a possible factor contributing to the resulting impairment in the light reflex. From this point of view the negative results obtained from eighteen cats having lesions largely confined to the thalamus make these cats valuable as controls.

It is not necessary to discuss in detail the results from each of the cats with thalamic lesions, and one cat may be selected as being representative of the group. Cat 22 (table 1) exhibited a complete loss of the light reflex two days after operation, but gradually improved so that fifteen days after the operation the light reflex was normal. During this period of transitory impairment in the light reflex, the pupils remained miotic, but regained their normal width on the day on which the normal light reflex was regained. A third peculiarity in the pupillary activity of this animal lay in the prolonged afterconstriction which

followed a calling into play of the light reflex. On cessation of illumination of the eye, the pupils remained constricted for a minute or longer, and only gradually regained their dilated state.

The lesion (fig. 2) lay in the caudal portion of the thalamus, immediately adjacent to the pretectal region, and destroyed the dorsal portion of the periventricular nucleus, the habenula, the dorsomedial portion of the nucleus lateralis posterior, the nucleus medialis dorsalis, the parafascicular nucleus and the *centre médian*, bilaterally. Only the most rostral edge of the pretectal region and posterior commissure were injured.

The results from this cat are representative of those from each of the cats with lesions in the caudal portion of the thalamus. Three

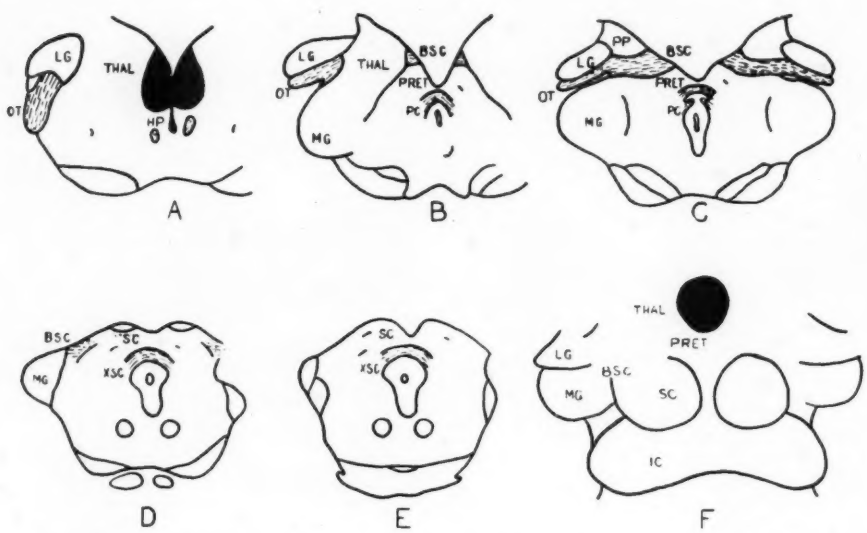

Fig. 2.—The location of the lesion in cat 22 is shown in solid black in five transverse levels extending from the caudal portion of the thalamus, *A*, through the pretectal region, *B* and *C*, to the rostral portion of the superior colliculus, *D* and *E*. Photographic representation of transverse sections through the cat's brain at the same levels have been published elsewhere (Ranson and Magoun[2]: fig. 1*A*, E; fig. 2*G*, *I* and *K*). The position of the lesion is shown also on the dorsal surface of the brain stem, *F*.

characteristic changes in the light reflex are associated with such lesions: (1) a transitory impairment, which becomes progressively less until, in from one to two weeks after operation, the normal light reflex is regained; (2) a transitory miosis of the pupil, which disappears with the recovery of the normal light reflex; and (3) a prolonged after-constriction of the pupil which follows cessation of illumination of the eye. A reasonable explanation suggests that the latter two symptoms are due to a transient irritation of the path of the light reflex by factors

associated with a lesion in its immediate vicinity.[1] The fact that such effects are produced by thalamic lesions indicates the proximity of the pathway of the light reflex to the caudal portion of the thalamus.

Bilateral Lesions of the Pretectal Region.—A study of the effect of bilateral lesions in the pretectal region has been made in eight cats. The size of these lesions varied in the different animals, and on this basis the series may be separated into three divisions: (1) animals with small lesions, (2) animals with lesions of intermediate size, and (3) animals with large lesions. The animals with lesions of small size may be described first.

Small bilateral lesions, destroying only the medial third to half of the pretectal region, were made in three cats, which were studied for from six to ten weeks after operation. Such lesions produced a complete loss of the light reflex on the day following operation, and at this time, but not subsequently, the pupils of these animals exhibited

TABLE 2.—*Cat 21, Operated on September 21*

Date	Width of Pupil Before Lighting		Lighting Right Eye		Lighting Left Eye	
	Right	Left	Direct	Consensual	Direct	Consensual
September 26	7	8	No reaction		No reaction	
October 26	10	9	No reaction?		No reaction?	
November 13	9	8.5	Trace	Trace	No reaction	
December 4	13	10.5	Trace	Trace	Trace	Trace
December 12	..	..	Bilateral cervical sympathectomy			
December 13	6.5	6	Fair	Fair	Fair	Fair

some miosis. Light reactions were recovered in the next few days, and became progressively better until a stabilization of impairment was reached at the end of about four weeks. Subsequent examinations up to the time at which the cat was put to death showed a fair light reflex, or a loss of about one-half the light reaction.

That this impairment was not due to the associated thalamic injury which occurred in each of these cats is shown by the observations which have just been reported on the effect of thalamic lesions. That the reduction in the light reflex could not be due to injury to the superior colliculus is evident from the fact that the damage to the rostral edge of the superior colliculus was negligible in two of the cats. It may be stated, therefore, that following bilateral destruction of the medial portion of the pretectal region, the light reactions from illuminating either eye are at first seriously impaired, but subsequently are restored to approximately one half of the normal extent.

The effect on the light reflex of pretectal lesions of intermediate size has been studied in two cats, one of which may be described in detail. Cat 21 (table 2) suffered a complete loss of the light reflex for

the first four weeks after operation. At that time a questionable trace of reaction in either pupil was observed under very strong lighting. Seven weeks after operation the right eye regained a definite trace of reaction to light, and ten weeks after operation a clear trace of reaction to light was regained by the left eye.

On the day before the animal was killed twelve weeks after the original operation, bilateral cervical sympathectomy was performed in order to observe the effect on the trace of light reaction of removal of the antagonistic, dilator innervation of the pupil. After cervical sympathectomy, the extent of the then unopposed constriction became augmented to almost one half of the normal light reflex, or to a fair reaction.

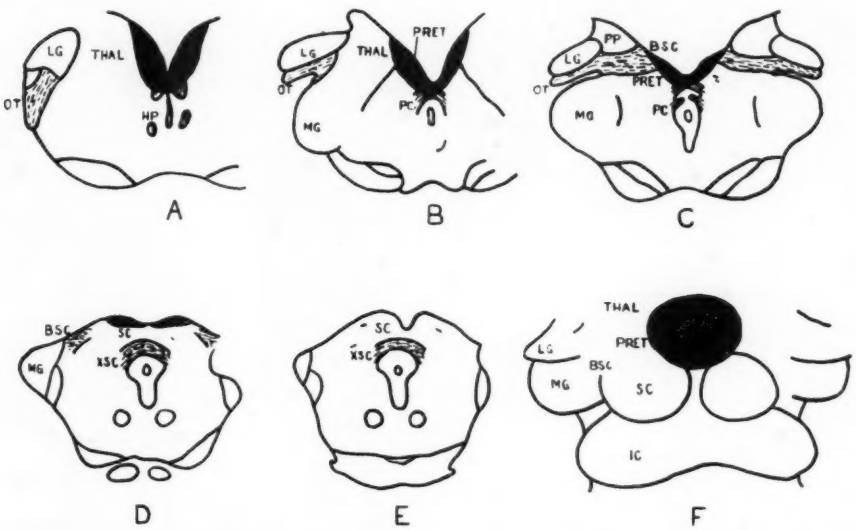

Fig. 3.—The location of the lesion in cat 21 is shown in solid black in five transverse levels extending from the caudal portion of the thalamus, *A*, through the pretectal region, *B* and *C*, to the rostral portion of the superior colliculus, *D* and *E*. The position of the lesion is shown also on the dorsal surface of the brain stem, *F*.

The lesion (figs. 3 and 7) injured the caudal portion of the thalamus, destroyed the medial half to two thirds of the pretectal region bilaterally and damaged a negligible amount of the rostral edge of the superior colliculus, bordering the intercollicular sulcus. The posterior commissure was partly interrupted.

The observations on the second cat with a pretectal lesion of intermediate size are almost identical with those just reported. The results from the two cats show that following bilateral destruction of the medial half to two thirds of the pretectal region, the light reflex is completely

abolished for the first month, but then a trace, or about one eighth, of the normal light reaction is regained. Following subsequent removal of the antagonistic, dilator innervation of the pupil, through bilateral cervical sympathectomy, the reaction is augmented to almost one half of the normal light reflex.

The effect on the light reflex of large bilateral pretectal lesions was studied in three cats, but the short survival time (three weeks) of one of these animals permits the data on it to be used only as supplementary. The other two cats may each be described in detail. Cat 8 (table 3) exhibited a complete loss of the light reflex for the entire duration of its survival, the animal being put to death seven weeks after the operation. On the day after operation, this animal exhibited severely miotic pupils, which measured 1 and 1.5 mm. in width, and showed no reaction to light. Two days after operation, the pupils had dilated somewhat,

TABLE 3.—*Cat 8, Operated on June 6*

Date	Width of Pupil Before Lighting		Lighting Right Eye		Lighting Left Eye	
	Right	Left	Direct	Consensual	Direct	Consensual
June 7	1	1.5	No reaction		No reaction	
June 8	4	3	No reaction		No reaction	
June 10	8	7	No reaction		No reaction	
July 6	8	6	No reaction		No reaction	
July 10	..	..	Bilateral cervical sympathectomy			
July 11	4	4	No reaction		No reaction	
July 12	6.5	6.5	No reaction		No reaction	
July 28	6	6.5	No reaction		No reaction	

measuring 3 and 4 mm. in width, and four days after operation the miosis had largely disappeared, and the pupils measured 7 and 8 mm. in width. An absolute loss of the light reflex was maintained, however, and no trace of recovery could be discerned in subsequent examinations.

One month after the original operation, bilateral cervical sympathectomy was performed in an effort to bring to view any latent trace of light reaction which might have survived. But on repeated testing after cervical sympathectomy, even when employing the intense light of a 60 watt bulb in a white reflector held immediately in front of the face, the pupils of this animal remained absolutely fixed and exhibited a complete loss of the light reflex.

On the day following sympathectomy the width of the pupils was reduced to 4 mm. from a previous width of 6 or 8 mm. On the second day after sympathectomy, the pupils had regained a width of 6.5 mm., which was maintained, even under intense lighting, for the subsequent period of survival. The animal was put to death three weeks after cervical sympathectomy, or seven weeks after the original operation.

The lesion (fig. 4) bilaterally destroyed the caudal part of the thalamus, the entire rostral portion of the pretectal region and the medial two thirds of the caudal portion of the pretectal region, together with the medial half of the rostral part of the superior colliculus. The entire posterior commissure was destroyed.

Cat 10 (table 4) also suffered complete loss of the light reflex for the entire duration of its survival, or for seven weeks following operation. Two days after operation this animal exhibited miotic pupils measuring 3 and 4 mm. in width, and showed a complete loss of the light reaction. Nine days after operation, the pupils were no longer miotic, measuring 9 mm. in width, but the absolute loss of the light reflex was maintained, and no subsequent recovery could be discerned.

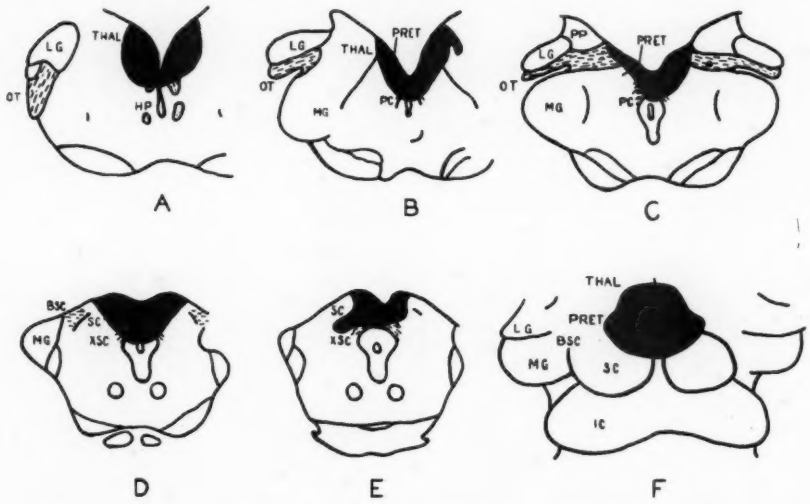

Fig. 4.—The location of the lesion in cat 8 is shown in solid black in five transverse levels extending from the caudal portion of the thalamus, *A*, through the pretectal region, *B* and *C*, to the rostral portion of the superior colliculus, *D* and *E*. The position of the lesion is shown also on the dorsal surface of the brain stem, *F*.

TABLE 4.—*Cat 10, Operated on June 8*

Date	Width of Pupil Before Lighting		Lighting Right Eye		Lighting Left Eye	
	Right	Left	Direct	Consensual	Direct	Consensual
June 10	3	4	No reaction		No reaction	
June 17	9	9	No reaction		No reaction	
July 19	7	7	No reaction		No reaction	
July 20	..	..	Bilateral cervical sympathectomy			
July 21	3	2.5	No reaction		No reaction	
July 26	6	6	No reaction		No reaction	
July 29	7	7	No reaction		No reaction	

Six weeks following the original operation, bilateral cervical sympathectomy was performed in an attempt, as in the previous case, to unmask any minute trace of the light reflex which might have survived. But in this animal also, on repeated testing, even when the intense light of a 60 watt bulb in a white reflector was employed, the pupils remained absolutely fixed.

On the day following cervical sympathectomy, the width of the pupils was reduced to 2.5 or 3 mm. from a previous width of 7 mm. Six days after sympathectomy, the pupils had regained a width of 6 mm., and a day later, a width of 7 mm., which was maintained even under intense lighting. The animal was put to death one week after cervical sympathectomy, or seven weeks after the original operation.

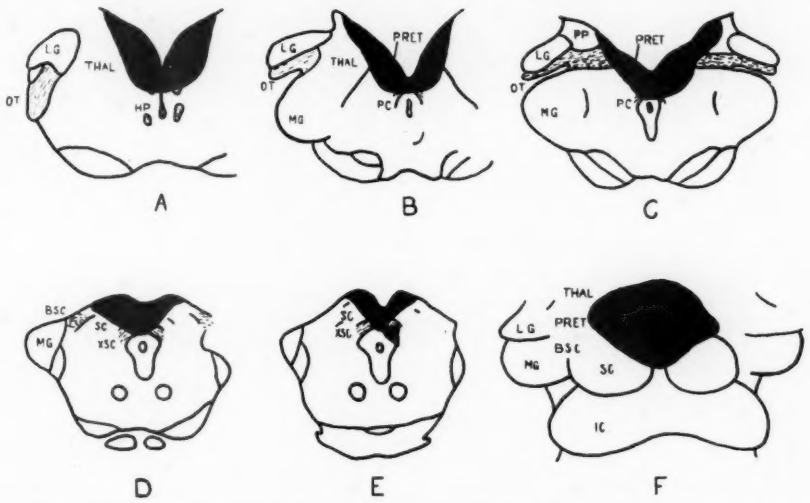

Fig. 5.—The location of the lesion in cat 10 is shown in solid black in five transverse levels extending from the caudal portion of the thalamus, *A*, through the pretectal region, *B* and *C*, to the rostral portion of the superior colliculus, *D* and *E*. The position of the lesion is shown also on the dorsal surface of the brain stem, *F*.

The lesion (fig. 5) bilaterally destroyed the caudal part of the thalamus, the entire rostral portion of the pretectal region and the medial two thirds of the caudal portion of the pretectal region, together with the medial half of the rostral part of the superior colliculus. The entire posterior commissure was destroyed.

All three of these animals with large bilateral pretectal lesions exhibited a complete loss of the light reflex. While one of the animals furnishes only complementary information, owing to its short survival time, the absolute loss of the light reaction in two of the cats was main-

tained even under strong lighting after bilateral cervical sympathectomy, the pupils under the conditions remaining moderately dilated. While the absolute loss of the light reflex in these animals is unquestionable, the large lesions in each case not only destroyed the pretectal region but injured also the rostral portion of the superior colliculus. This raises the question as to whether the damage to the rostral portion of the superior colliculus may be excluded as a factor contributing to the resulting abolition of the light reflex.

In considering the results from the three groups of cats in this series, which have been arranged according to the small, intermediate or large size of their pretectal lesions, it is apparent that a progressively greater reduction in the light reflex through the three groups goes hand in hand with an increasing damage to the pretectal region. Furthermore, the cats exhibiting a loss of all but a trace of the light reflex from pretectal lesions of intermediate size clearly indicate the relationship of the greater part of the pathway of the light reflex to the pretectal region.

In comparing, however, the cats losing all but a trace of the light reflex from pretectal lesions of intermediate size with those cats in which the light reflex had been completely abolished by larger lesions destroying more of the pretectal region, and materially injuring also the rostromedial portion of the superior colliculus, the complete abolition of the light reaction in the latter cats may be ascribed either to the greater destruction of the pretectal region or to the coincident injury to the rostral portion of the superior colliculus. While all of the evidence points to the conclusion that it is the damage to the pretectal region which is of significance, the point cannot be said to be unequivocally demonstrated by this material. The results from this series of animals should, therefore, be summarized in this way: While the possibility is not ruled out that a trace of the light reflex may be mediated by the rostromedial portion of the superior colliculus, it is clear that at least the greater part, if not all, of the light reaction is effected through the pretectal region.

Unilateral Lesions of the Pretectal Region.—The effect on the light reflex of unilateral lesions of the pretectal region was studied in a series of eight cats, which may be divided into two groups: The first group of three cats suffered unilateral destruction of the pretectal region, but the posterior commissure remained partly intact. In the second group of five cats, in addition to the unilateral damage to the pretectal region, the posterior commissure was destroyed up to or beyond the midline.

Cat 2 (table 5), representative of the first group, showed a complete loss of the light reflex on the day after operation, but a reaction was regained by the right eye three days following operation and by the left

eye on the fifth day. For the remainder of the survival time of eight weeks, tests of the light reflex in this animal revealed a marked asymmetry in the light reactions of the two eyes. While lighting the right eye, i. e., the eye on the same side as the lesion, produced a good direct and consensual constriction, lighting the left eye produced only a trace of reaction in either pupil.

Following bilateral cervical sympathectomy, performed six weeks after the original operation, the width of the pupils became somewhat less than before, and the light reaction was augmented, but the reaction from lighting the right eye still remained markedly greater than that from lighting the left eye.

The lesion (fig. 6) destroyed the caudal portion of the thalamus on the right side, the greater part of the right half of the pretectal region and a small amount of the rostral part of the superior colliculus on both sides of the midline. The entire left half of the pretectal region

TABLE 5.—*Cat 2. Operated on May 26*

Date	Width of Pupil Before Lighting		Lighting Right Eye		Lighting Left Eye	
	Right	Left	Direct	Consensual	Direct	Consensual
May 27	9	2	No reaction		No reaction	
May 29	5	6	Fair	Fair	No reaction	
June 10	6	7	Good	Good	Trace	Trace
July 10	..	..	Bilateral cervical sympathectomy			
July 28	4	5	Good	Good	Slight	Slight

and the greater part of the posterior commissure were not included in the lesion.

Two other cats, each possessing unilateral pretectal lesions which left the posterior commissure intact, resembled cat 2 closely. All three of these animals exhibited an impairment of the light reactions of the two eyes, but this impairment was markedly unequal.

The five cats belonging to the second group of this series exhibited essentially the same picture as that just reported. Coincident with the added destruction of the posterior commissure, however, the light reactions of the eye ipsilateral to the lesion showed a more severe impairment than in the case of the cats of the first group. This may be interpreted as favoring the view that the pathway of the light reflex undergoes a partial central decussation in the posterior commissure. Other evidence which supports this view will be presented elsewhere (Magoun, Ranson and Mayer [5]).

5. Magoun, H. W.; Ranson, S. W., and Mayer, L. L.: The Pupillary Light Reflex After Destruction of the Posterior Commissure in the Cat, Am. J. Ophth., to be published.

In each of the cats in this series, while the light reactions of both eyes have been impaired, the reduction has constantly been the more severe in the eye contralateral to the unilateral pretectal lesion. These observations indicate that while each half of the pretectal region receives impulses from both retinas those from the contralateral eye markedly predominate. This, in turn, suggests that the larger part of the pathway of the light reflex is crossed in the optic chiasma of the cat.

In the previous series of cats with bilateral lesions, it was observed that a progressively greater reduction in the light reflex, from cat to cat, went hand in hand with increasing bilateral damage to the pretectal region. In the present series of cats with unilateral pretectal lesions,

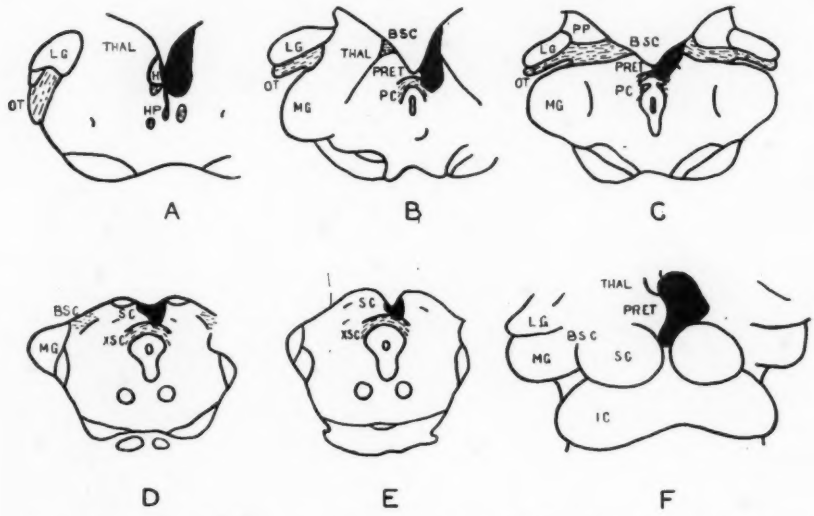

Fig. 6.—The location of the lesion in cat 2 is shown in solid black in five transverse levels extending from the caudal portion of the thalamus, *A*, through the pretectal region, *B* and *C*, to the rostral portion of the superior colliculus, *D* and *E*. The position of the lesion is shown also on the dorsal surface of the brain stem, *F*.

it has been seen, in comparing the light reflex impairment in the two eyes of an individual cat, that the more severe impairment of the light reflex of one eye may regularly be correlated with a greater damage to the contralateral half of the pretectal region. This direct variation of the impairment in the light reflex with the pretectal destruction, apparent under the varying conditions of the two series of experiments, confirms the results from electrical stimulation mentioned in the introduction to this paper, and demonstrates beyond question the relationship of the pathway of the light reflex to the pretectal region.

The relation of our observations on the pathway of the pupillary light reflex to the results obtained by other workers in this field will be discussed in a review of the literature appearing at a later date.

GENERAL OBSERVATIONS ON PUPILLARY ACTIVITY

Miosis.—The width of the pupils following lesions of the pathway of the light reflex is of interest in furnishing information bearing on the theoretical explanations of miosis of fixed pupils in man. The miosis of the Argyll Robertson pupil has been explained by Ingvar [6] as a stimulation of the light reflex fibers situated on the surface of the brain stem by chronic syphilitic meningitis.

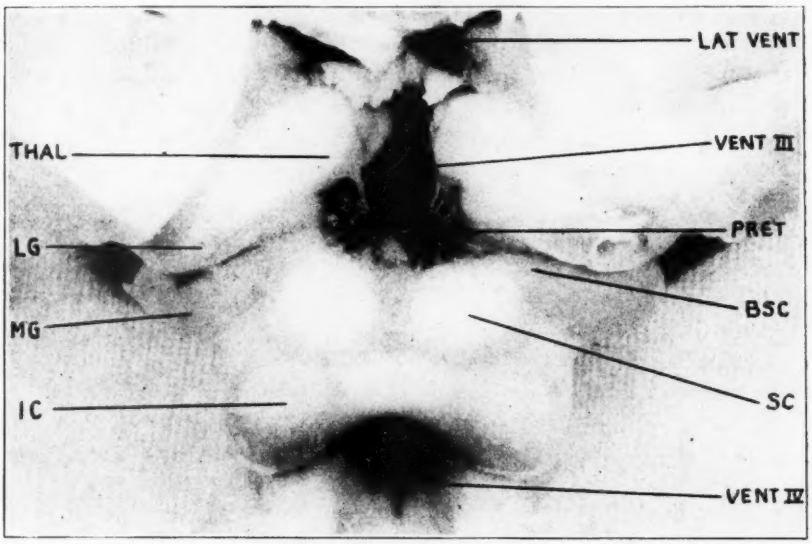

Fig. 7.—Dorsal view of the brain stem of cat 21. The pretectal region, or transition zone between thalamus and superior colliculus, is seen to be destroyed, while the lateral geniculate body, the brachium of the superior colliculus and the superior colliculus itself remain intact.

Effects on the pupil apparently due to chronic irritation of the path of the light reflex, though not by a meningitic process, have been encountered throughout these experiments. Following lesions of the caudal portion of the thalamus and the pretectal region, the pupils of the dimly lighted eyes may remain in a constricted state for as long as two weeks after operation. This miosis seems most reasonably to be ascribed to an irritation of the intact portion of the path of the light reflex by either mechanical or chemical agents related to the lesion.

6. Ingvar, Sven: On the Pathogenesis of the Argyll-Robertson Phenomenon, Bull. Johns Hopkins Hosp. **43**:363, 1928.

Two weeks was the longest period of time that miosis persisted in any of these experimental animals. The period was usually of a shorter duration. On the day after operation, the pupils were nearly always slitlike or markedly constricted. The constriction began to relax on the second day after operation, and after from three to five days the pupils had usually reached a steady state of dilatation.

This transient miosis was particularly marked in those cats in which the lesion was situated in the caudal portion of the thalamus, where it might be expected to irritate rather than to interrupt the pathway of the light reflex. Other miotic tendencies of the pupils of these animals with posterior thalamic lesions were noted. Their light reactions were characterized by persistent afterconstriction of the pupil following the cessation of lighting. At the cessation of illumination of the eye, an interval ranging between one and five minutes was required for the constriction of the pupil to relax, and for the original width of dilatation to be regained. This effect was not transient, however, but persisted for the survival time of the animal.

These observations demonstrate that the chronic irritation of a lesion in or near the path of the light reflex may produce miosis of a pupil for as long a period as two weeks, but they throw no light on the question whether such a process may be sustained over a period of years. Such a possibility seems improbable, unless the lesion is associated with a long lasting chronic irritation.

The miosis of the Argyll Robertson or fixed pupil of man was explained on a different basis by Merritt and Moore.[7] They emphasized the fact that the fixed pupil not only is miotic, but in addition fails to dilate to painful stimulation, and explained these symptoms on the basis of an interruption of both the pupillodilator and the pupilloconstrictor paths by a lesion within the mesencephalic tegmentum.

As we interpret Merritt and Moore, they regarded this as an interruption of the pupillodilator pathway which descends through the medulla to the lower cervical and upper thoracic portion of the spinal cord, to reach the pupil by way of the cervical sympathetic trunk. If this is the case, a peripheral destruction of the pathway in the cervical sympathetic trunk should have the same effect as a destruction in the midbrain. But, as has been reported earlier in this paper, in cats in which the light reflex had been abolished by pretectal lesions, an interruption of the pupillodilator pathway through bilateral cervical sympathectomy did not lead to miosis of the pupils. Under these conditions, the pupils exhibited slight miotic tendencies on the day after the sympathectomy, but subsequently remained moderately dilated.

7. Merritt, H. H., and Moore, M.: The Argyll Robertson Pupil: An Anatomic-Physiologic Explanation of the Phenomenon, with a Survey of Its Occurrence in Neurosyphilis, Arch. Neurol. & Psychiat. **30**:357 (Aug.) 1933.

The entire pupillodilator system was not abolished in these animals, however, for that portion of the path which acts centrally through an inhibition of the constrictor portion of the oculomotor nucleus still remained intact. This path was shown by Gozzano [8] to reach the oculomotor nucleus from its ventral side, and hence, as was pointed out by Spiegel,[9] would not be involved in a dorsally lying lesion destroying the afferent pathway of the light reflex.

Miosis of the pupils might be produced by destruction of both the oculomotor-inhibitory portion of the pupillodilator pathway and that portion which innervates the pupil by way of the cervical sympathetic trunk. Evidence which may be interpreted in favor of this was presented by Keller,[10] who observed a miotic pupil after transection of the brain stem through the caudal portion of the hypothalamus. Because of the short survival time of his preparations, the possibility is not ruled out, however, that this miosis was due to an irritation of a part of the pathway of the light reflex by the transection. In any case it should be emphasized that the phenomenon was produced by a transection of the brain stem and not by such a small lesion as might produce an Argyll Robertson pupil without other symptoms.

It is particularly difficult to account for a complete interruption of the pupillodilator pathway by a moderately sized lesion in the rostral part of the midbrain, because of the diffuse localization of the pathway at this level. The experimental observations from this laboratory (Ingram, Ranson and Hannett [11]; Ranson and Magoun [12]; Ranson, Kabat and Magoun [13]) demonstrated that in the rostral part of the midbrain marked bilateral dilatation of the pupils may be elicited from localized stimulation at almost any point within the extent of the mesencephalic tegmentum and the central gray matter of the aqueduct.

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8. Gozzano, M.: Wodurch kommt es zur Aufhebung der sogenannten sympathischen Pupillenreaktion bei bestehendem Argyll-Robertsonschen Phänomen? Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:644, 1925.

9. Spiegel, E. A.: Zur Lokalisation des Argyll-Robertsonschen Phänomens (insbesondere nach dem Verhalten der vestibulären Pupillenreaktion), Wien. klin. Wchnschr. **38**:189 and 216, 1925.

10. Keller, A. D.: Autonomic Discharges Elicited by Physiological Stimuli in Mid-Brain Preparations, Am. J. Physiol. **100**:576, 1932.

11. Ingram, W. R.; Ranson, S. W., and Hannett, F. I.: Pupillary Dilatation Produced by Direct Stimulation of the Tegmentum of the Brain Stem, Am. J. Physiol. **98**:687, 1931.

12. Ranson, S. W., and Magoun, H. W.: Respiratory and Pupillary Reactions Induced by Electrical Stimulation of the Hypothalamus, Arch. Neurol. & Psychiat. **29**:1179 (June) 1933.

13. Ranson, S. W.; Kabat, H., and Magoun, H. W.: Autonomic Responses to Electrical Stimulation of the Hypothalamus, Preoptic Region and Septum, Arch. Neurol. & Psychiat. **33**:467 (March) 1935.

These responses may be traced directly out of the hypothalamus, and we have no reason to doubt that they represent stimulation of an efferent system. If these results from the cat are applicable to man, a tremendous lesion would be required to interrupt the pupillodilator pathway at this level. Furthermore, such a lesion would have to be bilateral, since the pupils are bilaterally innervated from each half of the hypothalamus and midbrain in the cat and also in the monkey (unpublished observations of ours). The presence of unilateral miosis, therefore, remains unexplained.

But even though the pathway were somehow interrupted in the rostral midbrain, the question of the loss of pupillodilatation to pain still remains unsolved, for Keller [10] showed that after transection of the brain stem through the middle of the midbrain, caudal to the constrictor nuclei, a pupillodilatation to painful stimulation could still be elicited.

In conclusion, therefore, it may be stated that a number of experimental observations appear to be opposed to the suggestion that miosis of the pupils and loss of pupillodilatation to pain might be accounted for by interruption of the pupillodilator pathway by a moderately sized lesion in the rostral part of the midbrain.

Dilatation of the Pupil.—After section of the peripheral pathway for sympathetic impulses to the pupil, through bilateral cervical sympathectomy, pupillodilatation may still be produced by a central inhibition of the constrictor portion of the oculomotor nucleus. This inhibitory effect was found by Gozzano [8] to remain essentially intact after acute removal of the superior colliculus, together with extensive destruction of the central gray matter around the aqueduct. We were interested in determining whether the effect would remain after chronic lesions of the pretectal region which destroyed the afferent pathway of the light reflex, and thus interrupted for a period of weeks the main source of excitatory impulses to the sphincter portion of the oculomotor nucleus. Under these conditions would sufficient excitation be maintained in the sphincter portion of the oculomotor nucleus to permit an inhibition?

In two cats, after bilateral cervical sympathectomy, a dilatation of the pupils to painful stimulation was exhibited seven weeks after the light reflex had been completely and permanently abolished by bilateral lesions of the pretectal region. This indicates that even in the long-standing absence of light stimuli, some activity is still maintained in the sphincter nucleus. This activity seems to be produced by impulses reaching the nucleus from sources other than the retina. One type of constrictor activity which may be observed after abolition or impairment of the light reflex may now be reported.

Reflex Constriction of the Pupil from Causes Other Than Increased Intensity of Light.—In cats in which the light reactions had been either

abolished or impaired by pretectal lesions, a reflex constriction of the pupil, without changes in the illumination of the eye, could still be obtained by tapping the side of the nose or irritating the palpebral conjunctiva, by rapidly and forcefully opening and closing the lids, or by making threatening gestures, while holding the lids open to permit observations on the pupil. All of these actions induced an attempted blinking or lid closure, and the question arises as to whether we are dealing here with the orbicularis pupillary reaction which is found in man.

The orbicularis pupillary reaction of man was named by Gifford [14] who was one of the first to point out that a constriction of the pupil is associated with an attempt to close the lids. This reflex was described by Duke-Elder [15] as being entirely unilateral, with no consensual activity in the other pupil. Recently, Pollock[16] showed that by cocainization of the seventh nerve in the stylomastoid foramen the orbicularis reflex is abolished in man in spite of voluntary efforts to close the lids.

In the cat, this reflex constriction of the pupils persisted after bilateral section of the seventh nerves, with resulting abolition of the closure of the lids. It differed also from the lid closure reflex described in man, in always occurring bilaterally. This constriction may represent the oculosensory reflex the afferent impulses of which, according to Duke-Elder,[15] are mediated by the trigeminal nerve fibers innervating the eye and its vicinity. Opposed to this, however, is the fact that a constriction of the pupil was induced in some cats by visual stimulation of a threatening character.

This constrictor reflex could not be elicited on repeated testing in three cats with unilateral pretectal lesions, and in some of the tests a slight dilatation resulted from tapping the nose while holding the lids open. Four other cats with unilateral pretectal lesions exhibited good constrictor reflexes. Seven cats with bilateral pretectal lesions showed the reflex, but in three of these it was absent during the first weeks after operation, and in one case was not apparent until after cervical sympathectomy. Reduction of the antagonistic, dilator capacities of the pupil through cervical sympathectomy, in other cats in which this operation was performed, improved the constrictor reflex in these cats.

The amount of constriction of the pupil produced by this reflex in some cats with complete loss or severe impairment of the light reflex

14. Gifford, H.: An Orbicularis Pupillary Reaction, Arch. Ophth. **24**:402, 1895; The Palpebral Reaction of the Pupil (Galassi), ibid. **29**:191, 1900.

15. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, vol. 1

16. Pollock, L. J.: Personal communication, 1934.

almost equaled that of the normal light reaction. Before cervical sympathectomy, a reflex constriction of the pupil to 5 mm. from an initial width of 9 mm. was elicited in three cats, and a constriction to 3 mm. from a width of 9 mm. was obtained in three other cats. After cervical sympathectomy, a constriction of the pupil to 1.5 or 2 mm. was elicited in three cats.

While this response was normally associated with an attempted blinking or lid closure, it was induced in five of six cats after bilateral section of the seventh nerves, and showed no impairment under these conditions. The absence of lid movements in these cats permitted a clearer observation of any other phenomena which might accompany the reaction, and in three of the cats the constriction of the pupil was noted to be closely associated with a retraction of the eyeball, although in one cat a retraction of the bulb was observed without any accompanying constriction of the pupil.

In summary, following loss or impairment of the light reflex, a constriction of the pupils of the unlighted eyes may be produced by trigeminal or visual optic stimulation. While the reaction is normally accompanied by an attempted lid closure, it survives bilateral section of the seventh nerves. In some instances the reaction may be observed to be associated with retraction of the eyeball. This reflex constriction was not observed in normal cats, and appeared to increase with the severity and duration of interruption of the pupillary light reflex. A hippus of the pupil was frequently seen under the same conditions. It would appear that in the absence of its normal control by the afferent light reflex path, the sphincter portion of the oculomotor nucleus may react in an exaggerated manner to stimuli which normally would not cause noticeable constriction.

SUMMARY

A summary of the effects on the light reflex of lesions in the pretectal region of the brain may now be presented:

Lesions in the pretectal region of the cat's brain impair the pupillary light reflex. In the case of bilateral lesions the amount of reduction in the light reflex is roughly proportional to the amount of pretectal destruction. In the case of unilateral pretectal lesions the light reactions of the contralateral eye are the more severely impaired. If the pretectal region is almost completely destroyed on both sides of the brain, along with some damage to the rostromedial portion of the superior colliculus, the light reflex is completely and permanently abolished. While a minor participation in the light reaction on the part of the rostromedial portion of the superior colliculus cannot, therefore, be excluded, it is clear that at least the greater part of the pupillary light reflex is mediated through the pretectal region.

It should be emphasized that pretectal lesions in cats do not in any sense produce an Argyll Robertson pupil. No observable pupillary reaction to accommodation can be shown in the cat under either normal or experimental conditions. While these lesions cause a miosis lasting a few days, this disappears as the irritation caused by the lesion subsides. In the chronic stage, the pupils are widely dilated owing to the obliteration of the pupillary light reflex.

At the present time it is impossible to say whether the miosis seen in connection with the Argyll Robertson pupil is due to a chronic irritation of some part of the pupilloconstrictor path or whether it is due to an associated lesion in the pupillodilator pathway. From what we know of the courses followed by the pupillary light reflex and by the dilator path descending from the hypothalamus, it is difficult to see how both of these could be interrupted by a single lesion of moderate extent.

MIXED TUMOR OF THE ORBIT OF THE SALIVARY GLAND TYPE

SUCCESSFUL REMOVAL, WITH PRESERVATION OF EYEBALL

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NEW YORK

A case of mixed tumor of the orbit is reported because of the unusual character of the growth and also because of its occurrence with ocular signs due to pressure on the globe.

REPORT OF A CASE

P. K., a man, aged 32, was first seen on July 5, 1932. Eight months previously he became cognizant of the presence of ptosis of the upper lid of the right eye, which probably increased in the interim. His only other complaints were occasional blurring of vision, tearing and twitching of the lower lid of the right eye.

Fig. 1.—Photograph of the patient taken in 1931.

There had never been diplopia, metamorphopsia or pain. Glasses were first prescribed in 1924 and were not worn until they were changed in 1930. There was no history of trauma.

The positive findings were: The upper lid of the right eye was 3 mm. lower than that of the left eye, and there was retraction, together with some edema of the lower lid of this eye. With pressure directed backward on the right eye marked resistance was encountered. A mass could be palpated under the upper margin of the orbit. The right eye showed 21 mm. of exophthalmos, as compared with 16 mm. in the left (Hertel exophthalmometer). No bruit waas present, but the Wolff sign (altered breath sounds as heard with the stethoscope placed over the closed lids) was somewhat increased on the right side, as compared with the left. The right eye was pushed down as well as forward. The motility of the right eye was restricted on looking upward, especially to the right. Vertical diplopia (increased on looking upward) could be elicited only with the red glass. The corneal and conjunctival sensibility, the pupils, the fields and blindspots and the tension, as well as transillumination, were all normal. The fundus of the right eye presented an interesting picture. There were four or five parallel narrow retinal folds extending from the region about 0.5 diopter above the disk and running temporally toward the region above the macula. The diameter of each fold was about that of the large retinal arteries. This wrinkled area was slightly edematous,

but the retinal vessels could be clearly seen dipping in and out within the folds. Otherwise the fundus of each eye was perfectly normal. Vision in the right eye was 20/100; with correction it was 20/30 with a —4.50 cyl. ax. 5. Vision in the left eye was 20/20 with a —1.00 sph.

General physical examination showed no abnormality, except for the basal metabolic rate, which was —19 per cent. There were no evidences of congenital abnormalities, and the salivary glands apparently were normal. The blood count and differential blood count gave normal results; the hemoglobin was normal. The Wassermann test of the blood was negative. A roentgenogram of the right orbit was questionably cloudy.

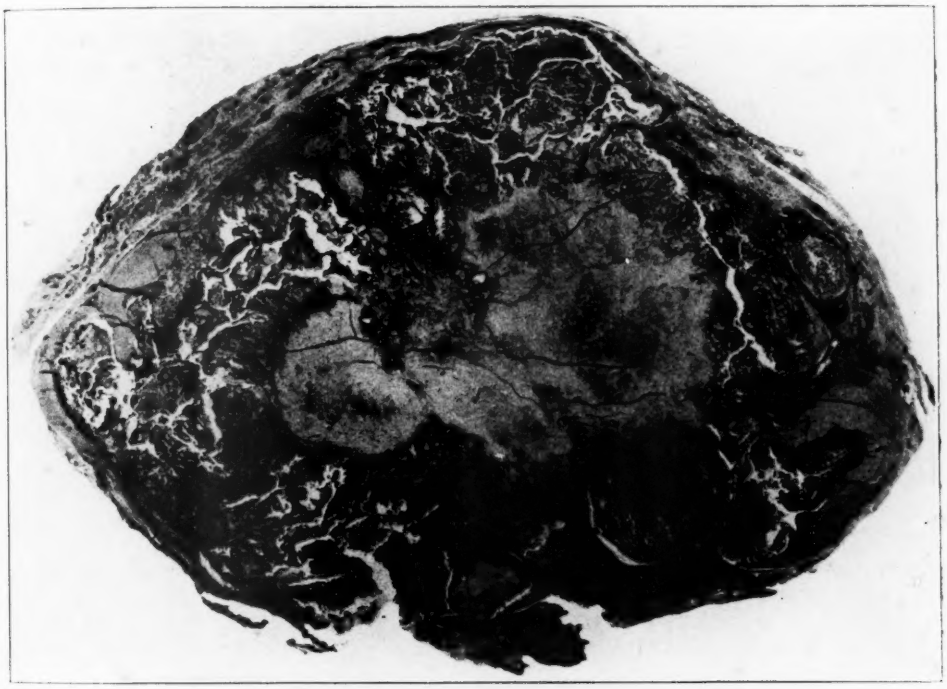

Fig. 2.—Photomicrograph of entire tumor; low power magnification.

Because of the possibility that the growth might be a radiosensitive neoplasm, such as giant cell follicular hyperplasia, or as a preoperative measure, the roentgen rays were applied in mild doses (applications that usually cause an apparent cure in giant cell lymphosarcoma) by Dr. A. Kean. No recession in any of the findings was noted.

A Krönlein resection was done on Feb. 9, 1933, by Dr. Arnold Knapp. After reflection of the osteoplastic flap through a tear in the orbital periosteum up and out, a bulging mass presented which appeared to be a tumor. This was about the size of a normal eyeball. It had a definite capsule and occupied the upper and posterior part of the orbit, being situated in a cavity within the soft parts, without any involvement of bone and without any particular adhesion. In its removal the

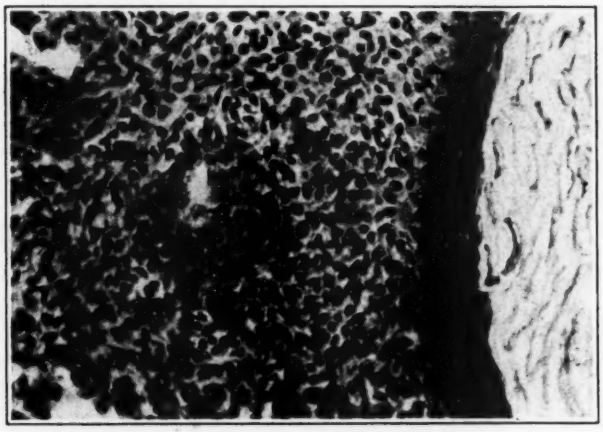

Fig. 3.—Section showing transition cell types, with basal cells, myxomatous degeneration and cornification.

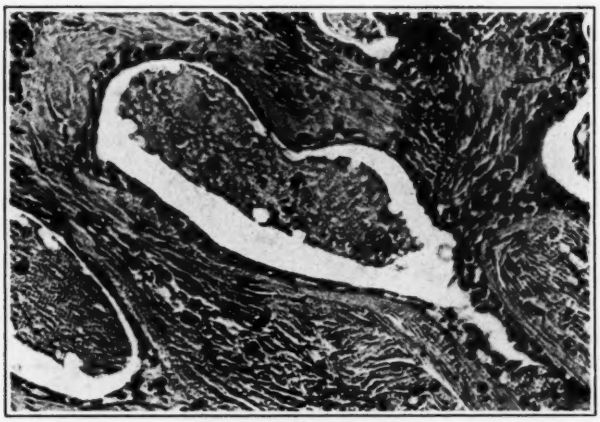

Fig. 4.—Section showing epithelial cells arranged in adenomatous and glandular formation.

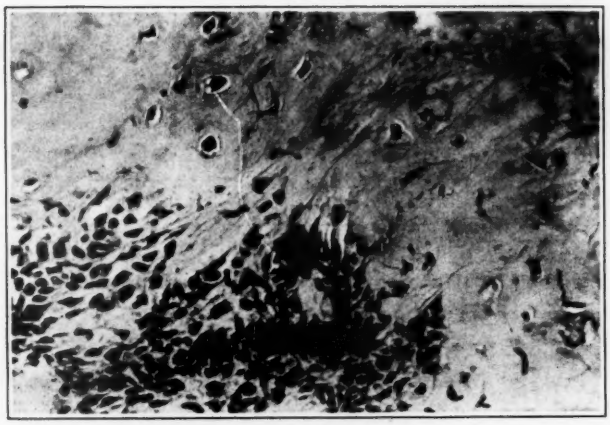

Fig. 5.—Section showing narrow zone of epithelial cells surrounding and fusing with precartilaginous and cartilaginous elements.

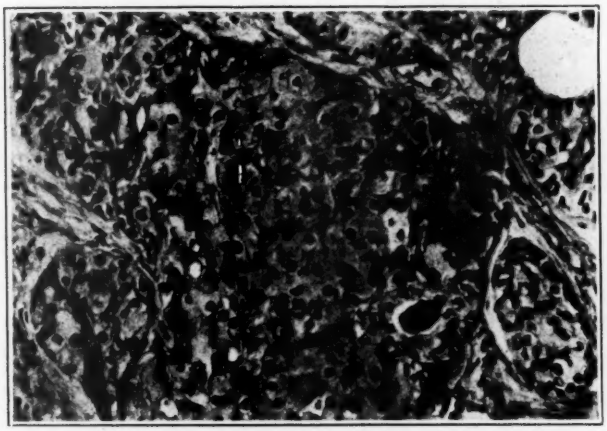

Fig. 6.—Section showing metaplastic cells.

tumor was broken into in one place, with the escape of a grayish granular material. The tumor was completely removed; the wound was closed, and sutures were applied through the eyelids.

Healing was uneventful, and on February 23 vision in the right eye was 20/30 with a —5.00 cyl. ax. 15. Right hyperphoria increased on looking up and to the right. The retinal lines were much less definite. On March 3, vision in the right eye was 20/30 with a —4.00 cyl. ax. 15. There was binocular single vision in all the lower fields. Exophthalmometric examination showed 17 mm. of exophthalmos in the right eye and 15 mm. in the left. All evidences of wrinkling of the retina disappeared after about three months. When the patient was last seen, in November 1934, vision in the right eye was 20/20— with a +0.50 sph. —3.00 cyl. ax. 5. The scars were well healed, and no recurrence was present.

Microscopically, the tumor was a typical mixed cell neoplasm of the parotid salivary gland type. A thick fibrous capsule was present at the periphery of the tumor mass, except where it was broken into at its removal. Strands of connective

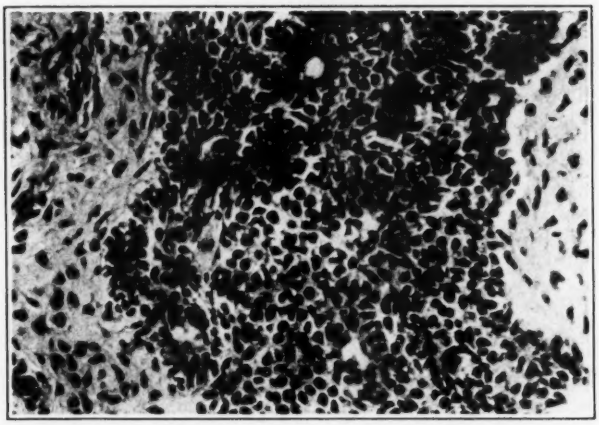

Fig. 7.—Section showing epithelial cells invading mucoid tissue.

tissue isolated large areas of compressed multicellular and, again, poorly cellular tissue. In each nest various types of cells were seen. Areas of round, spindle, stellate and metaplastic cells could be seen. Besides the solid masses of cells, there were also parenchymal areas with glandlike spaces having single or double rows of cuboidal epithelium. Myxomatous stroma containing precartilaginous and cartilaginous cells made up a great part of the tumor. There was an infrequency of mitosis.

COMMENT

A. It is possible that many of the tumors described as mixed tumors of the lacrimal gland may have had their origin from ectopic salivary gland fetal rests. The mixed tumors of this type apparently originate from rests which represent the anlagen of all three germinal layers, forming, however, from the unused blastomeres at a later stage than the more complex teratomas or dermoids. Though they may be distal to the parotid gland, these anlagen are referred to as parotid rests.

Mixed tumors derived from them or having their histologic make-up have been found in the palate, tonsillar region, trachea, sternoclavicular joint and skin and even involving the eyebrow,[1] and also the upper lid.[2] About 90 per cent appear about the region of the parotid gland.

Depending on the predominating components, these heterotypical tumors have been classified as adenocarcinoma, chondrosarcoma and other types. Usually all the cell types seen in mixed tumors of the parotid are present. They are more complex in the region of the parotid gland than elsewhere, according to Ewing.[3] In the extraparotid region they fall usually into the group of adenoid cystic epithelioma or cylindroma. In our patient the tumor was very complex. The mixed tumors of the caruncle are, as a rule, teratoids. The lacrimal gland is a serous type of gland and is classified with the parotid and submaxillary glands. Mathias [4] stated that in some species of animals (cats) salivary glands appear in the orbits and in rodents they are located near the tear sac.

In the review of many of the numerous cases of so-called mixed tumors of the lacrimal gland, from the descriptions and photomicrographic sections of the tumors described, I found that many were apparently isolated from the lacrimal gland. Some were even described as being definitely separated. Van Duyse [5] described a case in which, on the removal of the lacrimal gland for epiphora, he found a small nodular tumor of the mixed parotid gland type placed between and distinctly separated from both the accessory and the main lacrimal glands. Peters [6] grouped these tumors into "salivary gland mixed tumors of the region of the lacrimal gland."

B. Intra-orbital masses located about the globe may give rise to ocular compression phenomena. Among these are restricted motility of the globe, exophthalmos, involvement of the nerves of the bulb in their intra-orbital course, signs due to pressure on the optic nerve, macular pigmentary changes, detachment of the retina and changes in the axial refractive index. Astigmatic changes and also the formation of

1. Gerlach, G.: Mixed Tumor of the Eyebrow, Klin. Monatsbl. f. Augenh. **83**:807, 1929.

2. Puscariu, H.: Mischgeschwulst des Oberlides, Klin. Monatsbl. f. Augenh. **89**:493 (Oct.) 1932.

3. Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928, p. 768.

4. Mathias: Zur Lehre von den Prognonoblastomen, Virchows Arch. f. path. Anat. **236**:424, 1922.

5. Van Duyse: Tumeur mixte épithéliale de la région de la glande lacrymale, Arch. d'opht. **30**:401, 1910.

6. Peters, A.: Orbita, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1931, vol. 11, pt. 2, p. 478.

folds in the retina may occur. Knapp [7] described the occurrence of folds and pigmentary degeneration at the macula due to scleral indentation.

The corrugation folds noted in the patient were of a straight line type. They closely resembled the isolated, broken, linear retinal traction folds that are arranged circumferentially in the periphery of the retina soon after cauterization in some cases of detachment of the retina, where reattachment has taken place. In the latter cases they are distal to the area of cauterization in the uninvolved retina. The folds seen in the cases in which there was scleral indentation and the folds in the cases of detachment gradually disappeared in about three months after operative procedure. Momentary pressure exerted on the sclera with a glass rod apparently does not cause the formation of such folds.

Astigmatism due to bulbar compression is usually associated with myopic or hyperopic changes. Kubik [8] described a case in which 14 diopters of astigmatism was produced in this manner. The corneal and ciliary-lenticular distortion of the anterior segment probably accounts for at least some of the astigmatism. These changes probably are secondary to the posterior bulbar changes. There is a parallel example in the slightness of astigmatic changes in patients with high myopia in whom scleral ectasia also has played a part in the malformation of the globe. Kugelberg [9] reported astigmatic changes in a patient with a high degree of exophthalmos in exophthalmic goiter. I saw such changes in two patients with hyperthyroidism in whom only a moderate degree of exophthalmos was manifest.

1123 Park Avenue.

7. Knapp, A.: A Tumor (Cavernous Angioma) of the Orbit Causing Pressure Symptoms on the Eyeball, Arch. Ophth. **1**:704 (June) 1929.

8. Kubik, I.: Die Folgen der Bulbuskompression bei Raumbeengung in der Orbita, Klin. Monatsbl. f. Augenh. **80**:513 (April 27) 1928.

9. Kugelberg, I.: "Rillenbildung" in der Retina als Ausdruck einer Deformation des hinteren Bulbusabschnittes, Klin. Monatsbl. f. Augenh. **89**:171 (Aug.) 1932.

RETURN OF SYMPTOMS AFTER SUCCESSFUL REMOVAL OF A PITUITARY TUMOR

COMPLETE RESTORATION OF CENTRAL AND PERIMETRIC VISION BY MEDICAL TREATMENT

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The trend of present-day treatment of pituitary hyperplasia is undoubtedly surgical, for while roentgen irradiation and medical procedures have been and are still being employed and favorable reports from reliable sources have appeared from time to time, physicians seem reluctant to accept these results at their face value, to give them the serious consideration they seem to deserve and to encourage further research in this direction.

Certainly this attitude is not due to the infallibility of radical surgical measures, for although the mortality from either transphenoidal or transfrontal hypophysectomy is probably less than 5 per cent when these operations are performed by expert neurosurgeons, it is equally true that only 20 per cent of the patients retain useful vision more than five years after operation, and that of the remaining 80 per cent many either show no improvement after the operation or, following more or less marked improvement, have recurrences after two years.

In view of these facts and the indisputable long-standing favorable results reported from both roentgen ray and medical treatment, it would seem worth while to give these conservative measures a trial, especially in the early stages, before resorting to the more dangerous radical surgical intervention.

REPORT OF A CASE

The case reported is one of recurrent pituitary tumor. Operation was performed nearly seven years before writing, with complete restoration of central and peripheral vision. There was recurrence two years before writing, which was followed by a complete recovery of vision and general health under medical treatment as after the previous operation.

The patient was a married woman, 53 years old, with two adult children, one of whom was subnormal mentally. The family history was irrelevant. She had always been near-sighted and had worn strong minus lenses for twenty-five years. Her complaint was blindness in the right eye and partial blindness in the left eye.

In 1926 she had an attack of blindness involving both eyes, but especially the right eye. A diagnosis of pituitary tumor was made at that time, and she was successfully operated on by Dr. Howard C. Naffziger of the University of Cali-

fornia. Her condition before and after the operation can best be understood by the following extract from a personal communication from Dr. Naffziger, in which he stated:

"Mrs. R. came to me on Nov. 22, 1926, and was discharged from the hospital on December 6. . . . The trouble for which she entered gave clear evidence of its presence in January 1926, when she noted gradual failure of vision in the right field for the first time; since then the loss had progressed rapidly in both eyes, so that for the previous two months she had been unable to read or sew and was uncertain in walking. Headaches had increased in frequency. She had gained about 30 pounds (13.6 Kg.) during the year preceding April 1926. She had had either polyuria or frequency for many years, with no drowsiness, asthenia or feeling of cold and no increase in the intake of carbohydrate. At the time of admission there was complete temporal hemianopia in the right eye and limitation of the upper temporal quadrant in the left. Visual acuity in the left eye was 20/40, and in the right eye it was extremely bad. She was unable to read the chart when it was held 10 or even 5 feet (3.3 or 1.5 meters) from her right eye.

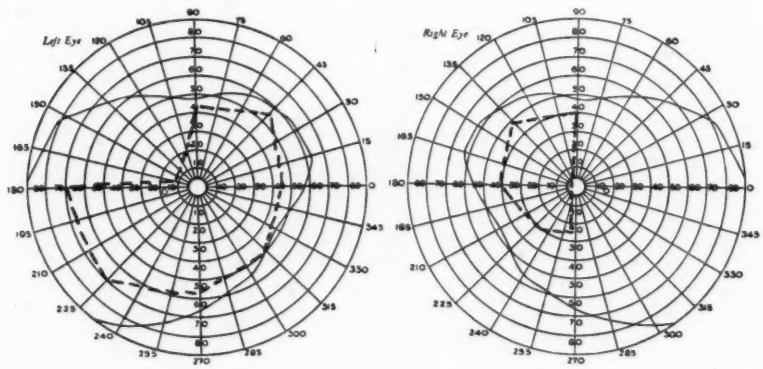

Fig. 1.—Visual fields taken on June 4, 1933, before beginning of treatment.

"Roentgenograms showed a marked enlargement of the sella turcica, with thinning and tipping backward of the posterior clinoid processes. The anterior clinoid processes were considerably spread apart, and somewhat eroded. The floor of the sella turcica seemed to show some erosion.

"A transphenoidal hypophysectomy was performed on November 26, and a subtotal removal of a chromophobe adenoma made. Convalescence was easy, and on December 6 she had full fields of vision to a 2 mm. disk in both eyes, vision of 20/20 in the left eye and 20/15 in the right. We were much pleased with her improvement."

Following the hypophysectomy she remained in good general health and had good vision until January, 1933, when vision in the right eye began failing. The condition grew progressively worse until June 4, when she came under my care. Shortly before that time vision in the left eye had also been failing, so that at the time of consultation she was unable to read or sew and had difficulty in walking about. Coincidentally with the visual decline, she had suffered frequent severe headaches in the occipital region, was unable to sleep in a reclining position and was compelled frequently to pace the floor during the night for the relief of the pain in the head.

The patient was rather tall, long-legged and short-bodied and had gained about 15 pounds (6.8 Kg.) during the previous six months, weighing 198 pounds (89.8 Kg.) at the time of examination. She was very weak, was unsteady on her feet and was unable to attend to her usual household duties. There was no history of drowsiness, increased intake of carbohydrate or polyuria since her previous operation. Both the Wassermann test of the blood and urinalysis gave negative results. She was wearing glasses with a —6.00 sph. ⌒ —1.00 cyl. ax. 90 for the right eye. Vision in the left eye was limited to counting fingers at 1 meter; with correction with a —4.50 sph. ⌒ —1.00 cyl. ax. 90 it was 5/7.5. A recent change in her glasses had failed either to relieve the headache or to improve the vision. Ophthalmoscopic examination of each eye showed that the media were clear and the fundus normal, except for slight hyperemia of the disks. In the visual field of the right eye there were complete temporal hemianopia, extending beyond the central fixation point, and considerable contraction of the nasal field, and in the left eye there was a large defect in the superior temporal quadrant.

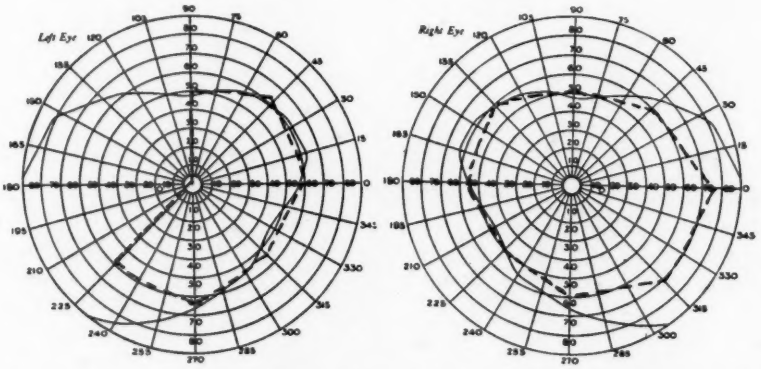

Fig. 2.—Visual fields taken on July 25, after seven weeks of treatment. Vision in the right eye has improved considerably, but that in the left shows further loss in the inferior temporal portion.

The roentgen examination showed pronounced enlargement of the sella turcica. A diagnosis of pituitary tumor, probably adenoma, was made.

The patient was advised of the possible need for another operation, which she greatly dreaded, but was encouraged to hope that something might be accomplished by means of medical treatment. A saturated solution of potassium iodide was prescribed, beginning with a dose of 5 drops (0.3 Gm.) three times a day, which was increased daily, and desiccated thyroid, 1 grain (0.06 Gm.), three times a day. One week later she reported having been unable to take the iodide because it caused headaches, pain in the back, nausea and vomiting. The thyroid caused no disturbance and was continued, and as a substitute for the potassium iodide the patient was given one tablet of lipoiodine Ciba night and morning. She did not begin taking the lipoiodine until June 20 on account of difficulty in obtaining this preparation in the small town in which she resided.

On June 23, her physician advised me by phone that a marked divergent squint had developed in the right eye, with dilatation of the pupil and diplopia.

When she was seen on June 25, the strabismus, dilatation and diplopia had all disappeared; the patient felt considerably better, and the headaches were lessened in frequency and severity. The visual fields were practically unchanged. Vision with correcting lenses was limited to the ability to count fingers at 1.5 meters in the right eye and was 5/7.5 in the left. At this time ophthalmoscopic examination showed definite papillitis in each eye. The dose of lipoiodine was increased to one tablet three times a day, and thyroid was continued as before.

On June 30, she reported by letter that her vision and general health were greatly improved.

On July 25, the headache had almost completely disappeared, but while vision in the right eye had improved, that in the left had diminished. Vision in the right eye was 5/6, and that in the left, 5/15, with correction. The right visual field was almost normal, but in the left most of the temporal portion was obliterated.

On August 10, vision in the right eye was 5/6, and that in the left, 5/9, with correction. Ophthalmoscopic examination of both eyes showed that the media

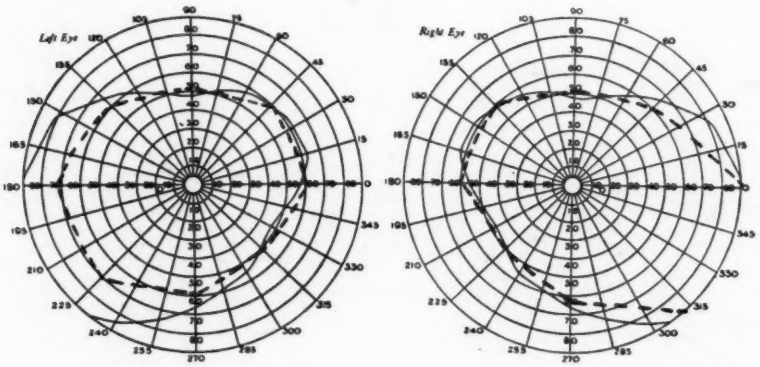

Fig. 3.—Visual fields taken on August 10, ten weeks after beginning of treatment.

were clear and the fundi normal. The right visual field was normal; the left extended to 70 degrees temporally.

Improvement continued uninterruptedly, and on December 5 the state of refraction was determined. With correction with a — 6.00 sph. ⌒ — 1.00 cyl. ax. 90, vision in the right eye was 5.5, and with correction with a — 5.50 sph. ⌒ — 1.00 cyl. ax. 75 vision in the left was 5.5.

Both fields were normal for form and colors. The patient's weight at this time was 183 pounds (83 Kg.). All medicine was discontinued. At the time of writing, eighteen months after treatment was instituted, the patient is in good health, entirely free from headaches and able to attend to all her household duties, and vision is normal in each eye with the correcting glasses.

What the future holds for her of course is altogether problematic. It can be stated, however, that medical treatment has, for the time being at least, given as satisfactory a result as could have been expected from the most successful operation, and if recurrence should occur, she will be advised to employ the same therapeutic measures, reenforced, if necessary, with roentgen ray treatment given by experts.

INTRACAPSULAR OPERATION FOR CATARACT

REPORT ON A FIFTH HUNDRED SUCCESSIVE EXTRACTIONS

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The series of operations reported on here comprises the fifth hundred successive intracapsular extractions of cataract. The report on the fourth hundred operations was published in the ARCHIVES [1] in July 1933. In presenting the present series, the final results are given, for though the vision at first is good, deterioration sometimes sets in later.

The cataracts were classified as to type as follows: mature, thirty-three; immature, twelve; nuclear, nine; nuclear and posterior cortical, twenty-seven; hypermature, seventeen, and morgagnian, two.

In thirteen cases preliminary iridectomy was done on account of the presence of a complication or in order to get information on the patient's behavior during operation. Another advantage of preliminary iridectomy is the absence of hemorrhage into the anterior chamber during the operation for cataract, which facilitates the application of the capsule forceps, though the formation of a satisfactory conjunctival flap may suffer and the patient is subjected to two operations. In these thirteen cases the cataracts were classified as follows: cataract associated with high myopia, one; complicated cataract, two; nuclear cataract, three; cataract in the only eye, four, and immature cataract, two.

The cataract was extracted by tumbling in eighty-nine cases; in seven of these the capsule ruptured during the delivery of the cataract and was extracted separately. A head-on delivery occurred in three cases, and in eight the lens was drawn out with the forceps remaining in situ.

The healing was complicated by prolapse of the iris in two cases, by hyphemia in nine and by loss of vitreous in five.

The resulting vision in fifty cases was 20/20; in five, 20/20 —; in twenty-two, 20/30, and in three, 20/30 —. This leaves twenty cases which will be described in detail.

In three cases in which the operation for cataract had progressed without complications, the patients died of lobar pneumonia, from bronchopneumonia and cerebral thrombosis and from coronary thrombosis, respectively. F. G. (case 70), aged 74, had pneumonia on the fourth day after operation and was transferred to the Roosevelt Hospital, where she died. A. G. (case 24), aged 76, had bronchopneumonia

1. Knapp, A.: Intracapsular Operation for Cataract: Report on a Fourth Hundred Successive Extractions, Arch. Ophth. **10**:6 (July) 1933.

on the fourth day after operation, from which she was apparently recovering, when hemiplegia set in, and death occurred as a result of the cerebral lesion. R. Z. (case 52), aged 61, died suddenly from coronary thrombosis on the fifth day after operation.

Respiratory complications in elderly patients are a serious menace to the operation. Perhaps with the intracapsular method the patient is kept even more quiet than after the usual extracapsular extraction, but it seems doubtful that this is the right practice, and it is more important for the patient to get up early, to do breathing exercises and to move the extremities. Possibly herein lies the advantage of the use of a sutured flap.

Of the remaining seventeen cases, the imperfect visual results in thirteen are explained by conditions which have no relation to the operation.

Case No.	Condition	Vision
2	Macula corneae; choroiditis; atrophy of the optic nerve	6/200
4	Old choroiditis	20/70
7	Chronic glaucoma; atrophy of the optic nerve	20/70
11	Chronic iridocyclitis; peripheric chorioretinal changes	20/20—
14	Tremulous iris; lens pulled out in bag; old detachment	8/200
16	Old detachment	Perception of light (?)
44	Atrophy of the optic nerve	20/200
50	Central choroiditis	6/200
62	Chronic glaucoma; atrophy of the optic nerve	20/40—
73 } 74 }	Retinitis pigmentosa; faulty projection	Perception of light (?)
80	Chronic glaucoma; atrophy of the optic nerve	20/50
85	Central choroiditis	20/50

The remaining four cases I wish to report in detail.

CASE 51.—*Slight loss of vitreous; detachment of retina two months later.*

A man, aged 60, had a posterior cortical cataract. The vision was 10/200. The eye was somewhat prominent and the cataract was sclerosed and immature. The operation was difficult because the usual anesthesia seemed insufficient. The patient became more and more nervous during the operation and had no control of his eyes. After the lens had been subluxated, the patient suddenly looked up, catching the incision in the speculum, with spontaneous expulsion of the nucleus and rupture of the capsule. The capsule was grasped with the forceps and slowly extracted, followed by a small escape of vitreous. The eye gradually recovered, and there was a faint hyaloid opacity drawn nasally to the incision. The vision with a correcting lens was 20/30. Two months later detachment of the retina suddenly occurred in the upper part of the fundus, and a tear could be seen in the periphery of the temporal region. A galvanocaustic operation was attempted for correction of the detachment of the retina, but it was not successful. This was a particularly difficult operation for cataract as it was done before the many admirable means that are now used for unruly patients were introduced, such as retro-ocular injection, suture of the superior rectus muscle and administration of

adequate sedatives, with possibly external canthotomy. The retinal detachment could not have come from the loss of vitreous, as the amount lost was very small. I am glad to say that the patient returned for operation on the second eye, and then with the use of the methods outlined the operation for cataract was done with an excellent result.

The remaining three cases exhibited the most serious complication which occurs in intracapsular operation: a clinical picture characterized by deep keratitis, with slow restoration of the anterior chamber, cyclitis and secondary glaucoma.

CASE 15.—*Difficult extraction; deep keratitis; cyclitis.*

A man, aged 57, had a posterior cortical cataract, with vision of 10/200. Syphilis had been acquired thirty years before, and the patient had reflex iridoplegia. At operation the delivery of the cataract was uncomplicated. The capsule had to be grasped twice with the blunt forceps to dislocate the lens definitely. Delivery of the lens was interrupted by its becoming jammed after presenting. Finally it was extracted after slight rotation with the cystitome. The difficulty was due to the lens being broad and flat. The operation was followed by congestion and a deep opacity of the cornea with wrinkling. This deep opacity of the cornea persisted, and deep blood vessels were formed. Tension was reduced. The vision with a correcting lens was 5/200. During an attack of increased tension an opening occurred in the line of the incision and the vitreous prolapsed. The iridocyclitis continued. The patient returned to his home and was lost from observation, and the operation was recorded as a failure.

CASE 59.—*Deep keratitis; cyclitis.*

The patient, aged 61, had a mature cataract in the right eye; intracapsular extraction was performed without complications. This was followed by a deep corneal opacity which partially cleared up; the vision with a correcting lens was at first 20/30. Then deep keratitis and opacities of the vitreous developed, and the sight became poorer. The keratitis progressed, and the ocular tension became soft. In brief, the case was one of deep keratitis and cyclitis with good preliminary vision which slowly proceeded to the loss of the eye. The extraction seemed to have been carried on without any particular complication, so it is hard to decide how much the operative maneuver was responsible for the outcome, unless there was some abnormality of the eye in addition to the cataract which made it incapable of withstanding this type of operation.

CASE 72.—*Difficult extraction; deep keratitis; cyclitis.*

The patient, aged 70, had a nuclear opacity with a posterior cortical opacity. The vision was 20/200. As the patient insisted on an operation, it was proceeded with. The lens, after prompt dislocation below, presented when pressure was applied, but it seemed to become wedged, and the cataract was extracted with difficulty though without apparent complications. A deep opacity of the cornea developed, and tearing was a prominent symptom, with moderate congestion. Nevertheless, the vision with a correcting glass at first equaled 20/30. The fundus showed optic neuritis with hemorrhages. Then gradually an opacity formed in the deeper layers of the cornea above with deep vessels, and one portion of the ciliary region above seemed particularly sensitive to palpation. More hemorrhages occurred in the fundus, and notwithstanding all forms of treatment, the corneal opacity extended downward, the eye continued to tear, and the ocular tension remained very soft. Vision

was gradually reduced to perception of light; the cornea showed deep opacities with blebs on its anterior surface, and there was a slight increase in tension; in brief, there was cyclitis which may have been the result of the trauma exerted in extracting the lens, of the many complications which the patient's general health presented or of a combination of the two factors.

It has always been a problem to me how much traction on the ciliary body the cataractous eye will stand. The resulting cyclitis in these cases cannot be ascribed to infection, so the cause must be traumatic. It is possible that in the lesser degrees of traumatism as produced with the cystitome or sharp-toothed forceps the eye will react with various degrees of iridocyclitis, in which, of course, tags of capsule and retained cortex play a rôle. On the other hand, certain eyes and certain patients with constitutional infirmities are undoubtedly more disposed than others. In such cases, and when the cataract does not subluxate on slight traction, it would be safer to desist from this procedure.

A discussion of the intracapsular operation must always take up the question of loss of vitreous. I have never been of the opinion that loss of vitreous is negligible; it is bound to prevent the prompt healing of the incision and lead to inflammatory reaction on the part of the eye, often showing itself in persistent opacities of the vitreous and bands. With this form of intracapsular operation, the loss of vitreous occurs after the delivery of the lens and so can be dealt with with little trauma, and the visual results are good.

In a few cases there seems to be an adhesion between the capsule and the hyaloid, so that the separation of these two structures does not take place without a rupture of the hyaloid membrane. The importance of this also varies according to the stage of the operation in which it occurs.

The five cases with loss of vitreous are the following:

CASE 8.—The capsule ruptured and was extracted separately, followed by slight prolapse of the vitreous. Recovery was uneventful. Vision was 20/30.

CASE 54.—The hyaloid membrane ruptured nasally with prolapse of the vitreous; the cataract was pulled out with the forceps without further loss. Vision was 20/30.

CASE 51.—The capsule ruptured and was extracted separately followed by a small escape of vitreous; detachment of the retina occurred two months later. Vision was limited to perception of hand movements.

CASE 94.—The hyaloid membrane ruptured below and temporally with prolapse of the vitreous; the capsule ruptured during delivery and was extracted separately with slight loss of vitreous. Vision was 20/30.

CASE 93.—When the nasal pillar was replaced after extraction, there was slight oozing of the vitreous. The incision was found closed; slow healing of the incision occurred under the flap. Vision was 20/20.

The hyaloid membrane ruptured in two cases before delivery of the lens. This complication may be due to the application of too much

force with the forceps, to the patient's unexpected movement or to an abnormal condition of the hyaloid membrane with adhesion to the suspensory ligament. When it occurs the anterior chamber always becomes deeper, and a gap forms between the iris and the cataract. The best treatment then, generally, after the speculum is replaced with retractors, is to grasp the cataract again with the blunt forceps and with counter-pressure below draw it out cautiously. In two cases in which the capsule was ruptured and was extracted separately, the process was followed by a prolapse of vitreous. In three cases the prolapse occurred after the extraction of the cataract and was not difficult to deal with. The rupture of the hyaloid membrane in these conditions presumably takes place at the same time when the capsule ruptures, and it proves that in all stages great gentleness and care must be exercised. In the last case of loss of vitreous slight oozing of vitreous followed the replacement of the nasal iris pillar; the healing was slow, but normal vision was obtained.

Loss of vitreous on completing the toilet of the incision occurred more frequently in my former series of cases; this complication is probably due to a deformation of the vitreous body after extraction of the cataract.

Iritis occurred in the following three cases:

CASE 7.—Iritis lasted two weeks, beginning on the sixth day without known cause. It did not influence the final vision (chronic glaucoma), which was 20/70.

CASE 54.—Loss of vitreous occurred; iritis developed on the sixteenth day but did not prevent good final vision, which was 20/30.

CASE 68.—The capsule ruptured during extraction; it was subsequently extracted separately. The patient had diabetes. Iritis lasted from the tenth to the twenty-fifth day after operation.

It is generally accepted that freedom from iritis is one of the great advantages of intracapsular operation and proves what an important factor retained cortex and capsule are. The iritis in the foregoing cases was mild and not troublesome. The severe form of cyclitis previously described in this report is quite a different manifestation and is the result of a serious lesion in the ciliary body.

Transient detachment of the choroid occurred in one case (case 42), in which final vision was 20/20.

The eyes with chronic glaucoma were in cases 7, 62 and 80. The intracapsular extraction was without complication; the tension did not return in two, but the use of miotics had to be continued in one.

The intracapsular operation practiced in this series is the same as that described in my previous papers, so the procedure need not be repeated. After trying other blunt capsule forceps, I have always come back to the use of the Kalt forceps, which I was the first to use for this

operation in 1910; recently I have been trying out the Arruga forceps with a great deal of satisfaction, and I shall continue to employ it.

Much has been written on the intracapsular operation during the past ten or fifteen years, as the practice of this method has been greatly extended, but no startling innovations have thus far resulted; merely small but important improvements have been introduced for the better control of the patient, such as anesthesia of the orbicularis muscle, suture of the superior rectus muscle, retrobulbar injection, external canthotomy, suture of the usual flap or of the sliding flap and suitable sedatives. All of these need not be applied in every case, but the operator should be prepared to use them.

LOCAL QUININE THERAPY IN CASES OF INTERSTITIAL KERATITIS AND OLD CORNEAL OPACITIES

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CHICAGO

In a paper read before the Chicago Ophthalmological Society on Nov. 19, 1934, I [1] reported the methods and results of local quinine therapy in trachoma. The rationale of the quinine therapy is that quinine, besides being a bactericide and an astringent, is a protoplasmic poison which penetrates deeply into the tissues when applied locally to mucous membranes. Binz,[2] Roth [3] and others showed that it destroys leukocytes and lymphocytes in and out of blood vessels, and that its destructive effect on lymphocytes is particularly marked. As Cushny [4] stated, quinine acts on the nutrition of almost all forms of protoplasm, causing first an augmentation of its activity, later depression and finally death. Experiments on animals and clinical trials in cases of trachoma showed that 2 per cent and 4 per cent quinine bisulphate ointment and a 10 per cent aqueous solution of quinine bisulphate can be applied locally daily for many months without any harmful effects on the conjunctiva, cornea and other ocular tissues.

This article is a preliminary report. The encouraging results of quinine therapy in cases of trachoma naturally suggested a trial of this drug in other conditions. In this communication I shall describe the results obtained in cases of interstitial keratitis and old corneal opacities resulting from interstitial keratitis and other causes.

INTERSTITIAL KERATITIS

It is the consensus that the course of interstitial keratitis cannot be influenced by the use of antisyphilitic or local therapy. Antisyphilitic treatment should, of course, be given to prevent the development of other manifestations of syphilis, but no reliance can be placed on its influence

From the Department of Ophthalmology of Rush Medical College, University of Chicago.

1. Selinger, E.: Studies in Protein Content of Human Aqueous; Influence of Therapeutic Measures; Significance in Papilledema, Papillitis and Other Conditions, Am. J. Ophth. **17**:1130 (Dec.) 1934.

2. Binz, C.: Arch. f. Mikros. Anat. **3**:383, 1867; Virchows Arch. f. path. Anat. **46**:67, 1869; **51**:6, 1870; Experimentelle Untersuchungen über das Wesen der Chininwirkung, Berlin, A. Hirschwald, 1868; Arch. f. exper. Path. u. Pharmakol. **1**:18, 1873; **5**:39, 1875; **7**:275, 1877.

3. Roth: J. Pharmacol. & Exper. Therap. **4**:157, 1912.

4. Cushny, A. R.: Text-Book of Pharmacology and Therapeutics, Philadelphia, Lea & Febiger, 1918.

on the inflammation of the eye. Local therapy in the form of atropine, hot dressings, dark glasses and other means of treatment is directed chiefly at the control of the accompanying inflammation of the iris, ciliary body and anterior portion of the choroid and to prevent, if possible, the development of complications.

In three cases of bilateral active interstitial keratitis of the type seen in congenital syphilis 2 per cent quinine bisulphate ointment was prescribed for use twice a day in addition to atropine, hot dressings and dark glasses. All three patients were receiving antisyphilitic treatment (neoarsphenamine and the newer arsenicals). Rapid subjective improvement was noted after a few days, so that the photophobia and blepharospasm, which were excessive before the use of the quinine ointment, were markedly diminished within a few days and the patients were able to keep their eyes open. Changes in the appearance of the corneal opacities were also observed. Old infiltrates became less dense and in some places disappeared entirely within from one to two weeks, while fresh corneal opacities, but of comparatively less density, made their appearance in other places from time to time. The appearance then was that of shifting deep corneal infiltration. No change was seen in the appearance of the newly formed corneal vessels. The entire course of the disease seemed to be milder and shorter. The beneficial effect of quinine in cases of interstitial keratitis is probably the result of several factors. The slight anesthetic effect of the quinine on the cornea may, in part, account for the rapid subjective improvement. Part of the subjective improvement, however, may be a result of the decrease in the severity of the inflammation. That the quinine has an effect on the accumulation of lymphocytes and other abnormal cells in the deep layers of the stroma is seen from the fact that the opacities are observed to change in density, and that some of the older infiltrates disappear entirely in places within a few days, while fresh, less dense infiltrates make their appearance from time to time. It is known that ordinarily corneal infiltrates, once developed, become denser and denser and remain for from many weeks to many months before they finally become absorbed. The rapid change in the character of the infiltrate can be ascribed only to the action of the quinine on the cellular elements making up the infiltrates. One of the three patients treated with the quinine bisulphate ointment showed improvement in central vision from 0.1 to 0.4 within seven days and to 0.8 within thirty days, although the inflammation continued for five more weeks. There had been no change in the medication, aside from the addition of the 2 per cent quinine bisulphate ointment.

What the bactericidal action of quinine on Spirochaetae pallidae in the corneal tissue is cannot be stated at this time. That the quinine does penetrate deeply into the tissues of the eye when applied locally

is shown by the fact that in a patient with old primary optic atrophy who was not taking any drugs by mouth I succeeded in obtaining a positive fluorescence reaction for quinine in 0.2 cc. of aqueous withdrawn from the anterior chamber after the instillation of 4 drops of a 4 per cent aqueous solution of quinine bisulphate into the conjunctival sac.

OLD CORNEAL OPACITIES

Equally interesting observations were made in cases of old corneal opacities after the use of 2 per cent quinine bisulphate ointment twice a day. Among seven eyes in four patients with old corneal opacities as a result of syphilitic interstitial keratitis, the condition was of twenty-four months' duration or longer in four, of eight months' duration in two and of five months' duration in one. These patients had been treated with ethylmorphine hydrochloride powder, 2 and 3 per cent yellow mercuric oxide ointment and hot dressings for varying lengths of time, with no changes in the appearance of the opacities and no improvement of vision. All other medication was discontinued when the treatment with the quinine was instituted. Within from two to six weeks most of the patients showed definite improvement of vision and a noticeable decrease in the density of the opacities. In one patient who had a dense central opacity of two years' duration, visual acuity of 0.4 improved to 0.8—2 after four weeks of local quinine therapy. There was still a great deal of clouding of the cornea left, but the center of the pupillary area had cleared sufficiently to increase the visual acuity to that extent. In another patient with a three year old very faint central opacity vision showed an improvement from 0.8 +3 to 1.0 + 3, while in a third patient vision which was only 0.2 as a result of an eighteen month old dense central opacity improved to 0.4—2. In some other cases there were extremely dense opacities, and although there was no measurable increase in the visual acuity there was a definite decrease in the density of the opacities, and the patients stated that objects appeared considerably brighter to them than before the treatment.

Three other patients had corneal opacities as a result of corneal ulceration. In one the condition was of three months', in another of eight months', and in the third of twelve months', duration. In the first patient visual acuity improved from 0.1 to 0.3, and in the second patient from 0.4 to 0.6, after three weeks of quinine therapy. The third patient had a very dense central opacity measuring about 6 mm. in diameter, which seemed to become less dense, but there was no improvement in visual acuity.

It is probably true that some of the opacities would have cleared up to a certain extent with other forms of therapy, or even spontaneously,

but the comparatively rapid improvement with the quinine therapy is striking. Two of the eyes with old interstitial opacities had shown no improvement after subconjunctival treatment with sodium iodide had been tried about one year before. Complete resorption of old opacities cannot be expected with any form of treatment, but it is reasonable to expect that some of the elements making up the corneal scars will be influenced by therapeutic measures. A slight improvement in visual acuity or even improvement in the cosmetic appearance makes it desirable to attempt any form of treatment that seems free from danger. Proper evaluation of the therapeutic action of a new drug can be obtained only after a sufficiently large number of patients have been treated over a long period of time. This report is submitted in the hope that other investigators may try this new therapeutic agent in suitable cases and report their findings. Further clinical studies, together with laboratory experiments, are being carried out to show the effect of quinine on these and other pathologic conditions of the anterior segment of the eye.

In cases of interstitial keratitis and old corneal opacities the 2 per cent ointment was used twice a day, just before retiring and again in the morning. The ointment causes a burning sensation which lasts for several minutes but becomes slight after the ointment has been used for several days. There is no cumulative action of the drug. The following drugs are among the antagonists and incompatibles of quinine and for that reason should not be used while the patients are being treated with quinine: copper, lead, zinc, mercury and their compounds, ammonia, alkalis, iodides and bromides and their salts, tannic acid and lime water. Among the synergists are iron, arsenic and mineral acids.

SUMMARY

Quinine, an alkaloid, besides being a bactericide and an astringent, is a protoplasmic poison which penetrates deeply into the tissues when applied locally to mucous membranes. It destroys leukocytes and lymphocytes and probably causes the absorption of abnormal tissue elements, such as newly formed connective tissue in the cornea. Quinine bisulphate ointment has a favorable influence on the course of interstitial keratitis and promotes clearing of old corneal opacities.

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ETIOLOGY OF EXOPHTHALMOS

CONSTITUTIONAL FACTORS, WITH PARTICULAR REFERENCE TO EXOPHTHALMIC GOITER

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Abnormal protrusion of the eyeball is encountered in a wide variety of both local and general conditions. This article is devoted to constitutional diseases in which exophthalmos appears as either a complication or a part of the general abnormality. A certain amount of overlapping exists, because much of the exophthalmos which is encountered in general disease depends on the local accumulation in the orbit of tumors, hematomas or other masses, on the production of stasis in the orbital veins or on edema of the orbital tissues, all of which may result in forward dislocation of the eyeball. This generalization at times applies to the exophthalmos of exophthalmic goiter, which will be considered presently. It is partly, often wholly, applicable to the exophthalmos that occurs occasionally in other constitutional abnormalities.

EXOPHTHALMOS IN DISEASES OTHER THAN EXOPHTHALMIC GOITER

Exophthalmos is rare in syphilis. Delord [1] described bilateral protrusion of the eyes that occurred in the clinical course of a lesion of the pituitary body which was probably syphilitic, and Löhlein [2] wrote of paretic exophthalmos that appears in the course of syphilis or tabes, in which paralysis of the external muscles of the eye occurs. This is never of high grade and is accounted for by the loss of restraining action of the external ocular muscles. A gumma may involve the orbit, and there may be a tubercle of the orbit in tuberculosis; other granulomas may occur in a similar manner. Key [3] recently reported

From the Division of Medicine, the Mayo Clinic.
Read before the American Academy of Ophthalmology and Otolaryngology, Chicago, Sept. 10, 1934.

1. Delord, Emile: Sur un cas d'exophthalmie bilatérale considérable par lésions de la région hypophysaire; guérison par le traitement mercuriel associé à la radiothérapie profonde, Arch. d'opth. **49**:799 (Dec.) 1932.

2. Löhlein, Walther: Repetitorium der Augenheilkunde: VI. Exophthalmus und Enophthalmus, Deutsche med. Wchnschr. **56**:663 (April 18) 1930.

3. Key, B. W.: A Case of Trichinosis with Exophthalmos, Am. J. Ophth. **12**:178 (March) 1929.

a case in which exophthalmos accompanied trichinosis, and Holström [4] described the cases of two sisters with exophthalmos of the right eye which occurred in association with neurofibromatosis (Recklinghausen's disease). A few cases of this condition have been seen by Benedict [5] at the Mayo Clinic.

Exophthalmos is encountered also in cases of multiple xanthomatosis. This is a generalized disease in which tumorous masses of reticular cells are engorged with lipids, predominantly cholesterol. Occasionally the condition is associated with diabetes insipidus and defects of the membranous bones. It then constitutes the so-called Schüller-Christian syndrome. Multiple xanthomatosis is rare, and exophthalmos is by no means an invariable accompaniment of it. We have recognized only one instance of xanthomatosis with exophthalmos.

The various forms of leukemia sometimes are responsible for proptosis, but Giffin, with his large experience in diseases of the blood, could tell us of no single instance at the clinic, and the literature contains little about the condition. The only lymphoid tissue in the orbit, according to Reese and Guy,[6] is situated under the bulbar conjunctiva and in the lacrimal gland. The bulging of the eye in a case of lymphatic leukemia that was described by these authors was the result of swelling of the conjunctiva from the accumulation beneath it of a mass of lymphocytes. Exophthalmos in myelocytic leukemia is almost unknown. In the one case that was seen by Reese and Guy enucleation of the eyeball disclosed that the orbit was filled with blood. An instance of exophthalmos which occurred in monocytic leukemia was recently reported by Mesropyan.[7]

Hemorrhage in the orbit is the cause of the proptosis that is an occasional complication of hemophilia, rickets and scurvy. According to Löhlein, bilateral subperiosteal hematoma in the orbit, with resulting exophthalmos, for a long time may be the only manifestation of rickets. Rainess [8] wrote that approximately 2, 000 children, aged from 2 weeks to 2 years, make 15,000 calls yearly to the infirmary in the Russian city Astrakhan; in 1928, 620 of 2,020 children were obviously rachitic, and 120 of these had exophthalmos. There was a positive correlation between this symptom and the severity of the rickets, and with the disappearance of the rickets the exophthalmos disappeared. The exophthalmos of rickets differs from that of exophthalmic goiter

4. Holström, M.: Zwei Fälle von Exophthalmus bei Neurofibromatosis Recklinghausen, Acta ophth. **6**:403, 1928.

5. Benedict, W. L.: Personal communication to the authors.

6. Reese, A. B., and Guy, Loren: Exophthalmos in Leukemia, Am. J. Ophth. **16**:718 (Aug.) 1933.

7. Mesropyan: Bilateral Exophthalmos in Monocytic Leukemia, Sovet. vestnik oftal. **1**:558, 1932.

8. Rainess, L.: Eye Symptoms in Rachitis, Arch. Pediat. **47**:55 (Jan.) 1930.

in that in the former condition the lower lid is edematous and hangs down in the middle, and the upper lid, although also swollen, is not retracted. It may be mentioned in this connection that Yudkin, Farquhar and Wakeman [9] obtained exophthalmos, among other abnormalities, in rats that had been fed diets low in mineral elements.

Protrusion of the eyeball was discovered in nearly 10 per cent of 379 cases of scurvy that were investigated by the American Pediatric Society in 1898. This is cited from an article published in 1931 by Dunnington,[10] who commented, however, that De Buys, in 1912, had been able to discover only 12 reported cases of scurvy in which the evidence justified, in his opinion, the diagnosis of proptosis. To these 12 cases Dunnington added 10 more, including 1 of his own. He explained the condition of the eye in all instances on the basis of hemorrhage, usually subperiosteal, of the roof of the orbit. The protrusion of the eyeball is forward, downward and outward; it is of varying intensity and is usually a late manifestation. The average age of patients is 22 months, but Krukow [11] recorded the case of an adult, seen in Russia, who had scurvy and exophthalmos. The exophthalmos recedes rapidly as a result of treatment of the scurvy.

The occurrence of exophthalmos in association with hypertension and nephritis is a subject to which we have devoted considerable attention since it was reported by Barker and Hanes [12] in 1909. Their conclusions, which were supported by Thayer,[13] left the impression that it was a frequent complication of this condition. It should be noted that many of their patients were Negroes, a race in which prominent eyes are not unusual; that no exophthalmometer readings were reported, and that the possibility of the coexistence of exophthalmic goiter may not have been excluded as completely as it would be today. Our experience has not been the same. The determination of the presence or absence of exophthalmos and the estimation of the degree of proptosis in exophthalmic goiter, as well as the differential diagnosis of true exophthalmos and of conditions which simulate it, have been among our major problems for many years. It is our opinion, as well as that of H. S.

9. Yudkin, A. M.; Farquhar, Lucille, and Wakeman, A. J.: Some Abnormalities in Rats Subsisting on Diets Poor in Mineral Nutrients, Arch. Path. **17**: 40 (Jan.) 1934.

10. Dunnington, J. H.: Exophthalmos in Infantile Scurvy, Arch. Ophth. **6**: 731 (Nov.) 1931.

11. Krukow, cited by Dunnington.[10]

12. Barker, L. F., and Hanes, F. M.: Exophthalmos and Other Eye Signs in Chronic Nephritis, Am. J. M. Sc. **138**:469 (Oct.) 1909.

13. Thayer, W. S., in discussion on Barker, L. F., and Hanes, F. M.: Exophthalmos and Other Eye Signs in Chronic Nephritis, Tr. A. Am. Physicians **24**: 165, 1909.

Plummer [14] and his other associates, that retraction of the eyelids and possibly other factors which may contribute to a facial expression somewhat like that observed in exophthalmic goiter are not infrequent in nephritis but that the occurrence of proptosis is extremely rare. The difficulty of distinguishing an appearance of the eyes resulting from exophthalmos from that produced simply by retraction of the lids is exemplified in certain cases of exophthalmic goiter: One eye seems to be more prominent than the other, but the exophthalmometer readings are identical, and the apparent prominence is entirely attributable to retraction of the lids.

Confusion on this score seems to be responsible for most of the diagnoses of exophthalmos that are reported; this is true not only in hypertension and nephritis but in other conditions of so-called sympathicotonia. Similarly, the coexistence of exophthalmic goiter must frequently have escaped recognition. Conditions of mild hyperthyroidism are easily overlooked, and patients who have exophthalmic goiter and who have no evidence of hyperthyroidism at the time of observation are frequently seen; under these circumstances the condition is difficult to recognize. This has been emphasized by H. S. Plummer.

EXOPHTHALMOS IN EXOPHTHALMIC GOITER

Exophthalmic goiter, in which there is associated diffuse hyperplasia of the parenchymal epithelium of the thyroid gland, is the one constitutional condition in which exophthalmos is truly significant. Therefore, conclusions regarding the etiology and mechanism of the exophthalmos of constitutional diseases must depend primarily on the information that is accumulated by the study of this disease.

Experimental Exophthalmos.—In 1858 and 1859, Heinrich Müller [15] described the presence of smooth muscle in the eyelids. Landström [16] stated that as late as 1901 no smooth muscle had been recognized within the orbit and that Merkel and Kallius, who wrote in that year for the Graefe-Saemisch "Handbuch," insisted that the muscles of Müller extended only from the orbital margin to the tarsus and thus were in no way able by their contraction to influence the position of the bulb. Landström, with serial sections of entire orbits in three perpendicular planes, demonstrated a cone of smooth muscle that extended from the orbital margins to a position behind the equator of the eyeball. He attributed to this muscle the rôle of an antagonist to the four rectus muscles, by which the axis of the eye is given proper stability. The

14. Plummer, H. S.: Functions of the Normal and Abnormal Thyroid Gland; Histology of the Normal Thyroid, in Christian, H. A., and Mackenzie, J.: Oxford Medicine, New York, Oxford University Press, 1920, vol. 3, p. 846.

15. Müller, Heinrich, quoted by Landström.[16]

16. Landström, John: Ueber Morbus basedowii; eine chirurgische und anatomische Studie, Stockholm, P. A. Norstedt & Söner, 1907.

observation on animals, originating with Claude Bernard, that stimulation of the cervical sympathetic nerves causes forward displacement of the eyeball Landström considered to be explained by the presence of this cone of smooth muscle. He emphasized that these muscle fibers are overlooked if the eye is enucleated, because they then remain behind in the orbit. Serial sections of the entire orbit are necessary for their demonstration.

MacCallum and Cornell [17] likewise discovered that the smooth muscle fibers of Müller extend backward as a conical mantle about the eye. While they are more fully developed in the dog, they are by no means wanting in man. These investigators removed the roof of the orbit in dogs and by electrically stimulating the cervical sympathetic nerves they could observe a distinct peristaltic wave, which passed backward through the length of this cone and pressed the eyeball forward into the position in which it is seen in exophthalmos. When the membranous cone was snipped with scissors and the cervical sympathetic nerves were stimulated, the eyeball remained stationary. Jonnesco reported the production of exophthalmos at surgical operation by stimulating the sympathetic nerves with a strong current, but in 2 patients whom MacCallum and Cornell were able to observe through the courtesy of the surgical staff of Johns Hopkins Hospital stimulation of the cervical sympathetic nerves did not produce visible exophthalmos, although the pupil became greatly dilated on the stimulated side.

This anatomic and experimental evidence relative to the influence of Müller's or Landström's muscle was accepted [18] as a satisfactory explanation of the mechanism of exophthalmos by the author of an editorial that appeared recently. The complexity of the clinical problem was ignored. No such mechanism can possibly produce the more extreme grades of proptosis which are encountered in some cases of exophthalmic goiter, and surgeons who are acquainted with the anatomy of the human eye are almost universally of the opinion that the smooth muscle fibers of the orbit are developed too poorly in many, if not in most, instances to affect the position of the eyeball. Furthermore, widening of the pupil always accompanies artificial stimulation of the cervical sympathetic nerves and is seen clinically in irritative lesions of the neck, whereas this is unusual in the exophthalmic eye in exophthalmic goiter. The careful measurements of the pupils in a small series of cases which were reported by Holloway, Fry and Wentworth [19] conform to this conclusion.

17. MacCallum, W. G., and Cornell, W. B.: On Mechanism of Exophthalmos, M. News **85**:732 (Oct. 15) 1904.

18. The Mechanism of Exophthalmos, Editorial, J. A. M. A. **102**:2188 (June 30) 1934.

19. Holloway, T. B.; Fry, W. E., and Wentworth, H. A.: Ocular Signs in One Hundred Unselected Cases of Goiter, J. A. M. A. **92**:35 (Jan. 5) 1929.

Possibly the absence of mydriasis in the exophthalmos of exophthalmic goiter has been explained by the recent studies of Labbé and his associates.[20] Briefly, their results were as follows: Ephedrine when injected into dogs in doses of 0.5 mg. per kilogram produced mydriasis and exophthalmos that were scarcely noticeable, whereas if thyroxin was administered either before or afterward, intense exophthalmos developed without mydriasis. There was no increase in intra-ocular tension, no engorgement of vessels and no edema or retrobulbar congestion.

Epinephrine, when administered with thyroxin, had a like effect. This we may add, was noted also by Kendall and Haines [21] in early unpublished experiments with thyroxine after its isolation by Kendall.

The following clinical experiments were described by Labbé:

> An obese woman was receiving a large dose of thyroxin by mouth (10 mg. a day), and marked signs of hyperthyroidism were evident. The patient was given 50 mg. of ephedrine; five minutes later striking exophthalmos appeared, which was more marked in the left eye. It attained its maximum intensity in ten minutes and persisted for two hours. It was not accompanied by the slightest degree of mydriasis. Subsequently, two patients who had exophthalmic goiter but no exophthalmos and who had basal metabolic rates of about + 60 per cent, were given ephedrine, and exophthalmos developed and attained a degree of considerable importance within an hour and a half. This likewise was unaccompanied by mydriasis.

Exophthalmos accompanied by miosis was produced in animals with various combinations of miotic poisons and sympathomimetic drugs, and the assumption was made that thyroxine not only stimulates the sympathetic nerves but exerts a miotic action by simultaneous stimulation of the parasympathetic mechanism, by which its mydriatic effects are neutralized.

These observations, if confirmed, will constitute an important contribution. It is unfortunate that there is no statement in these reports regarding actual measurements of the degrees of exophthalmos. It should be mentioned that Labbé and his associates also discovered that yohimbine is antagonistic to ephedrine and epinephrine and tends to counteract the exophthalmos produced in animals by the latter drugs and that in some cases it exerts a favorable influence on the exophthalmos of exophthalmic goiter.

Authors who have experimented with animals have been unable to provoke exophthalmos with thyroxin alone, even when the doses were sufficiently large to produce tachycardia and loss of weight. However,

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20. Labbé, Marcel; Villaret, Maurice; Justin-Besançon, L., and Schiff-Wertheimer: Recherches sur la pathogéne de l'exophthalmie Basedowienne et son traitement, Bull. Acad. de méd., Paris **110**:730 (Dec.) 1933; Bull. et mém. Soc. méd. d. hôp. de Paris **47**:1883 and 1897 (Dec. 21) 1931.

21. Kendall, L. C., and Haines, S. F.: Personal communication to the authors.

Kunde [22] reported positive results with this procedure. Dogs, she wrote, are refractory to thyroxin, but in normal rabbits mild exophthalmos is obtained, while in rabbits which previously have been rendered myxedematous by thyroidectomy it develops to a high degree.

Exophthalmos has also been produced in animals by the injection of extracts of the anterior lobe of the pituitary body. It is now well established that ablation of the pituitary body causes atrophy of the thyroid gland and a significant decrease in the basal metabolic rate and that the injection of extracts of the anterior lobe of the pituitary body stimulates temporarily the activity of the normal thyroid gland. The latter phenomenon is accompanied invariably by hypertrophy of the parenchymal epithelium of the thyroid gland. The injections are also followed by exophthalmos, as was noted first in ducks by Schochaert [23] and as was noted later in guinea-pigs by Loeb and Friedman,[24] Marine and Rosen [25] and Friedgood.[26] Marine and Rosen expressed the opinion that exophthalmos could be produced even in the absence of the thyroid gland. In Friedgood's experiment, in 9 of 30 guinea-pigs that were given injections of extract of the anterior lobe of the pituitary body exophthalmos of obvious degree developed; at one time or another this exophthalmos was essentially unilateral. When it was bilateral one eye was frequently more prominent than the other.

A feature of the hyperthyroidism that is produced by repeated daily injections of extracts of the anterior lobe of the pituitary body is its transiency. Loss of weight and other evidence of increased activity of the thyroid gland appear for ten or fourteen days, and after this the animal enters a refractory stage of apparent immunity. In a few of Friedgood's animals the exophthalmos was observed by the tenth day, that is, during the height of the hyperthyroidism; in others it did not appear until after the refractory stage had been reached. The most

22. Kunde, M. M.: Studies on Metabolism: VI. Experimental Hyperthyroidism, Am. J. Physiol. **82**:195 (Sept.) 1927.

23. Schochaert, J. A.: Enlargement and Hyperplasia of the Thyroids in the Young Duck from the Injection of Anterior Pituitary, Am. J. Anat. **49**:379 (Jan.) 1932.

24. Loeb, L., and Friedman, H.: Exophthalmos Produced by Injections of Acid Extract of Anterior Pituitary Gland of Cattle, Proc. Soc. Exper. Biol. & Med. **29**:648 (Feb.) 1932.

25. Marine, D., and Rosen, S. H.: Exophthalmos in Thyroidectomized Guinea-Pigs by Thyrotopic Substance of Anterior Pituitary, and Mechanism Involved, Proc. Soc. Exper. Biol. & Med. **30**:901 (April) 1933.

26. Friedgood, H. B.: The Ocular Manifestations of Sympathetic Nervous System; Hyperactivity in Conditions Other Than Exophthalmic Goiter and Especially in Essential Hypertension; "Lidspasm": A New Eye Sign, Am. J. M. Sc. **180**:836 (Dec.) 1930; Experimental Exophthalmos and Hyperthyroidism in Guinea-Pigs: Clinical Course and Pathology, Bull. Johns Hopkins Hosp. **54**:48 (Jan.) 1932.

striking exophthalmos was seen in animals that were given injections over a period of several months, especially those in which abnormally low basal metabolic rates finally were obtained. Chronic exophthalmos resulted in some cases.

The last-mentioned instances of exophthalmos are extremely interesting; they are analogous, as will appear, to the exophthalmos that occurs in cases of remission of exophthalmic goiter. The most severe ocular complications that lead to so-called malignant exophthalmos in exophthalmic goiter develop after thyroidectomy has been performed or after spontaneous remission of the symptoms, when the basal metabolic rate has fallen to normal or even to less than normal. However, the evidence deduced from experiments on animals must be applied to man with the greatest caution. Exophthalmic goiter does not develop spontaneously in animals, as it does in man, and in one's enthusiasm over the experimental production of exophthalmos one must not disregard the tremendous amount of evidence provided by clinical experience. One fact, above all others, must be held in mind, namely, that resection of the thyroid gland in the vast majority of instances of exophthalmic goiter brings about not only subsidence of the symptoms of hyperthyroidism but also recession, or at least a standstill, of any previous exophthalmos. This demonstrates that the process which is responsible for the development of exophthalmos in this disease is in most cases markedly interfered with, if not entirely inhibited, by removing the influence of the thyroid gland. Only exceptionally does exophthalmos progress when other evidence of overactivity of the thyroid gland has disappeared, and too much emphasis should not be placed on these exceptions. The vast amount of clinical evidence still indicates that the immediate cause of exophthalmos in cases of exophthalmic goiter is closely linked with actual functional and pathologic changes in the thyroid gland.

Clinical Observations.—This brings us to the clinical data at hand. The incidence of exophthalmos in exophthalmic goiter has been variously reported by different authors. In Joll's [27] series it was 74 per cent; Boothby [28] reported it to be 65 per cent, and Barker [29] estimated it at only 34 per cent. We have reviewed the records of 2,000 patients at the clinic in an attempt to correlate the presence or absence of exophthalmos and its degree with the duration of the disease and with certain clinical features which are believed to be related to the severity of the disease and its duration. These are, namely, the basal metabolic rate

27. Joll, C. A.: Diseases of the Thyroid Gland with Special Reference to Thyrotoxicosis, St. Louis, C. V. Mosby Company, 1932.

28. Boothby, W. M.: The Thyroid Problem, Collected Papers of the Mayo Clinic, Philadelphia, W. B. Saunders Company, 1928, vol. 20, p. 495.

29. Barker, L. F.: The Diagnosis of Exophthalmic Goiter, J. A. M. A. **49:** 1235 (Oct. 12) 1907.

and the loss of strength of the quadriceps femoris muscles. H. S. Plummer called attention to the variations in the intensity of exophthalmic goiter that occur from time to time, at least in certain localities; periods of increased severity may last a year or more and alternate with periods of relative mildness. Also, a large percentage of patients who have exophthalmic goiter and who have been seen in recent years, have been treated with iodine, and this has modified the severity of the symptoms and has lowered the basal metabolic rate of those who have been treated just previous to coming under observation. Consequently, it seemed advisable to study two groups of cases removed from each other in point of time: 1,000 which were seen before 1922 and an equal number

TABLE 1.—*Basal Metabolic Rates of Patients with Exophthalmic Goiter* *

	Before 1922		After 1930		Total	
Basal Metabolic Rate	Number of Patients	Percentage of Patients	Number of Patients	Percentage of Patients	Number of Patients	Percentage of Patients
−30 to −20	...	....	1	0.1	1	0.05
−20 to −10	...	....	...	....	...	....
−10 to 0	...	....	3	0.3	3	0.15
0 to 10	5	0.5	14	1.4	19	0.95
10 to 20	31	3.1	79	7.9	110	5.50
20 to 30	91	9.1	181	18.1	272	13.60
30 to 40	156	15.6	203	20.3	359	17.95
40 to 50	166	16.6	169	16.9	335	16.75
50 to 60	193	19.3	167	16.7	360	18.00
60 to 70	131	13.1	94	9.4	225	11.25
70 to 80	107	10.7	51	5.1	158	7.90
80 to 90	64	6.4	23	2.3	87	4.35
90 to 100	39	3.9	10	1.0	49	2.45
100 to 110	11	1.1	3	0.3	14	0.70
110 to 120	1	0.1	2	0.2	3	0.15
120 to 130	1	0.1	..	...	1	0.05
130 to 140	2	0.2	..	...	2	0.10
140+	2	0.2	..	...	2	0.10
Total	1,000	100.0	1,000	100.0	2,000	100.00
Mean basal metabolic rate	+53.5		+42.4		48.0	

* The mean basal metabolic rate was lower in the patients seen after 1930 than in those seen before 1922.

which were seen after 1930. The cases in each group are consecutive, but only those of patients who had previously undergone thyroidectomy are included; the diagnosis of exophthalmic goiter, therefore, has been established not only by the clinical evidence but by direct examination of the thyroid gland and by the pathologic observation of diffuse hypertrophy of the parenchymal epithelium. Some of the thyroid glands contained adenomas, but no cases of the clinical entity known as adenomatous goiter with hyperthyroidism have been included. Exophthalmos is not an accompaniment of adenomatous goiter with hyperthyroidism. For all practical purposes, it is limited exclusively to the entity known as exophthalmic goiter.

Table 1 contains the basal metabolic rates. It reveals that the average severity of hyperthyroidism was relatively lower for the later group of cases (+ 42.4) than for the earlier group (+ 53.5).

Table 2 gives the distribution of patients by age and by sex. The mean age was slightly higher in the later period. This is true for both males and females. The proportion of males was appreciably higher in the later group.

TABLE 2.—*Distribution of Patients by Age and Sex* *

	Prior to 1922			After 1930			Total		
	Number of Patients	Percentage	Mean Age, Years	Number of Patients	Percentage	Mean Age, Years	Number of Patients	Percentage	Mean Age, Years
Males	225	22.5	39.3	374	37.4	42.7	599	30.0	41.4
Females	775	77.5	35.0	626	62.6	40.4	1,401	70.0	37.4
Total	1,000	100.0	36.0	1,000	100.0	41.3	2,000	100.0	38.6
Sex ratio, female to male		3.4			1.7			2.3	

* The mean age of patients who had exophthalmic goiter was higher for those seen after 1930 than for those seen before 1922. This is true for both males and females. The ratio of females to males was greater in those seen in the early period (3.4) than in those seen in the later period (1.7).

TABLE 3.—*Relation of Basal Metabolic Rate to Exophthalmos in Two Thousand Cases* *

	Degree of Exophthalmos											
	Absent		Grade 1		Grade 2		Grade 3		Grade 4		Present but Not Graded	
Basal Metabolic Rate	Number of Patients	Percentage	Number of Patients	Percentage	Number of Patients	Percentage	Number of Patients	Percentage	Number of Patients	Percentage	Number of Patients	Percentage
−30 to −20	...	....	...	....	1	0.3	...	....	...	....	...	....
−20 to −10	...	....	...	....	...	....	...	....	...	....	...	....
−10 to 0	2	0.2	...	....	1	0.3	...	....	...	....	...	....
0 to 10	8	0.9	3	0.6	4	1.1	3	2.9	...	....	1	1.3
10 to 20	54	5.9	22	4.2	20	5.4	5	4.9	1	11.1	7	8.9
20 to 30	155	17.1	68	13.0	35	9.5	4	3.9	1	11.1	8	10.1
30 to 40	195	21.5	78	14.9	54	14.6	20	19.6	...	....	11	13.9
40 to 50	158	17.4	93	17.8	52	14.1	17	16.7	2	22.2	12	15.2
50 to 60	145	16.0	110	21.0	66	17.9	18	17.6	3	33.3	16	20.2
60 to 70	103	11.3	64	12.2	40	10.8	10	9.8	1	11.1	7	8.9
70 to 80	55	6.1	42	8.0	39	10.6	13	12.7	...	....	8	10.1
80 to 90	16	1.8	24	4.6	31	8.4	8	7.8	...	....	6	7.6
90 to 100	9	1.0	11	2.1	21	5.7	4	3.9	1	11.1	3	3.8
100 to 110	6	0.7	6	1.1	2	0.5	...	....	...	....	...	....
110 to 120	2	0.2	..	...	1	0.3	...	....	...	....	...	....
120 to 130	..	...	..	...	1	0.3	...	....	...	....	...	....
130 to 140	..	...	2	0.4	...	....	...	....	...	....	...	....
140 +	1	0.1	..	...	1	0.3	...	....	...	....	...	....
Total	909	100.0	523	100.0	369	100.0	102	100.0	9	100.0	79	100.0
Av. basal metabolic rate	+44.1		+50.0		+53.1		+51.5		+50.4		+49.8	

* There appears to be a positive correlation between the basal metabolic rate and the relative incidence of exophthalmos. For patients who had no exophthalmos the average basal metabolic rate was +44.1; for those who had exophthalmos in various degrees the basal metabolic rate was +50 or more.

Table 3 reveals a positive correlation of the basal metabolic rate and the relative incidence of exophthalmos. For patients who had no exophthalmos the average basal metabolic rate was + 44; for those who had various degrees of exophthalmos the basal metabolic rate was + 50 or more.

The percentage of frequency of exophthalmos in exophthalmic goiter was definitely greater in the cases seen before 1922 than in those seen after 1930 (table 4). This correlates with the lower basal metabolic rates that were discovered in the later group. It is apparent for all grades of exophthalmos (table 5).[30] In other words, in the group in which the basal metabolic rates were lower the incidence of exophthalmos was also lower.

Table 6 reveals a positive correlation between the duration of hyperthyroidism and the relative frequency of exophthalmos. In the entire

TABLE 4.—*Percentage of Frequency of Exophthalmos in Exophthalmic Goiter**

	Before 1922		After 1930		Total	
	Number of Patients	Percentage of Patients	Number of Patients	Percentage of Patients	Number of Patients	Percentage of Patients
Exophthalmos	689	68.9	402	40.2	1,091	54.5
No exophthalmos	311	31.1	598	59.8	909	45.5
Total	1,000	100.0	1,000	100.0	2,000	100.00

* The relative percentage of exophthalmos in exophthalmic goiter was greater in the patients seen before 1922 than in those seen after 1930. Almost 70 per cent of the earlier group had exophthalmos; only 40 per cent of the later group had exophthalmos.

TABLE 5.—*Percentage of Frequency of Types of Exophthalmos in Exophthalmic Goiter* *

	Before 1922		After 1930		Total	
Degree of Exophthalmos	Number of Patients	Percentage of Patients	Number of Patients	Percentage of Patients	Number of Patients	Percentage of Patients
None	311	31.1	598	59.8	909	45.5
Grade 1	337	33.7	186	18.6	523	26.2
Grade 2	225	22.5	144	14.4	369	18.4
Grade 3	78	7.8	24	2.4	102	5.1
Grade 4	5	0.5	4	0.4	9	0.4
Present, but not graded	38	3.8	41	4.1	79	4.0
In left eye only	6	0.6	3	0.3	9	0.4
Total	1,000	100.0	1,000	100.0	2,000	100.00

* The greater percentage of frequency of exophthalmos in patients seen in the early period is revealed for all grades of exophthalmos.

2,000 cases of exophthalmic goiter there was an incidence of exophthalmos of 48.7 per cent in the cases of less than six months' duration, and this rose steadily to about 60 per cent for the cases having a duration of from two to four years and to 71.2 per cent for cases of more than four years' duration.

30. The grading of exophthalmos in these cases was based on inspection alone. The examiners, however, were physicians who had been trained to distinguish true prominence of the bulb from apparent prominence resulting from retraction of the eyelids. There is no reason to believe that the percentage of error in series as large as these would change; therefore, the grading that was done after 1930 can be assumed to be the same as the grading done before 1922.

In the period before 1922 the percentage of frequency of exophthalmos in the cases showing no loss of strength of the quadriceps femoris muscles was 62.7 per cent (table 7); this frequency increased steadily to 77.6 per cent as one went through the groups up to grade 5 loss of strength. In the period after 1930 the percentage of frequency in the cases showing no loss of strength was 33.8 per cent, and this rose to 44.7 per cent in cases with grade 5 of loss of strength.

TABLE 6.—*Relation of Duration of Constitutional Symptoms of Exophthalmic Goiter to Exophthalmos* *

	Prior to 1922			After 1930			Total		
		Exophthalmos			Exophthalmos			Exophthalmos	
Duration of Constitutional Symptoms	Number of Patients	Number of Cases	Percentage	Number of Patients	Number of Cases	Percentage	Number of Patients	Number of Cases	Percentage
5 mos.	241	164	68.0	262	81	30.9	503	245	48.7
6 to 11 mos.	196	136	69.4	166	73	44.0	362	209	57.7
1 to 2 yrs.	195	128	65.6	136	67	49.3	331	195	58.9
2 to 3 yrs.	85	58	68.2	68	35	51.5	153	93	60.8
3 to 4 yrs.	36	28	77.8	30	11	36.7	66	39	59.1
4 yrs. +	87	68	78.2	69	43	62.3	156	111	71.2
Not stated	160	107	66.8	269	92	34.2	429	199	46.4
Total	1,000	689	68.9	1,000	402	40.2	2,000	1,091	54.6

* There is a positive correlation indicated between the duration of the constitutional symptoms of exophthalmic goiter and the percentage of frequency of exophthalmos.

TABLE 7.—*Relation of Loss of Strength of Quadriceps Femoris Muscles to Exophthalmos* *

	Prior to 1922			After 1930			Total		
		Exophthalmos			Exophthalmos			Exophthalmos	
Loss of Strength of Quadriceps Femoris Muscles	Number of Patients	Number of Cases	Percentage	Number of Patients	Number of Cases	Percentage	Number of Patients	Number of Cases	Percentage
None	83	52	62.7	142	48	33.8	225	100	44.4
Grades 1 and 2	424	289	68.1	465	184	39.6	889	473	53.2
Grade 3	178	130	73.0	104	50	48.1	282	180	63.8
Grades 4 and 5	58	45	77.6	67	30	44.7	125	75	60.0
Not stated	256	173	67.6	210	86	41.0	466	259	55.6
Present but not graded	1	0	0	12	4	33.3	13	4	30.8
Total	1,000	689	68.9	1,000	402	40.2	2,000	1,091	54.6

* There is a definite positive correlation between the degree of the loss of strength of the quadriceps femoris muscles and the presence of exophthalmos.

This table also indicates that exophthalmos had developed earlier in the course of the disease among the patients who had been examined before 1922 than among those who had been examined after 1930.

Loss of strength of the quadriceps femoris muscles, as estimated by the ability of the patient to step up on the step of the examining table, had been recorded for many years. Table 7 discloses a positive correlation between the degree of the loss of strength of the quadriceps femoris muscles and the presence of exophthalmos.

The bearing of this correlation on the mechanism of exophthalmos is to be considered. It rests on the assumption that loss of strength of the quadriceps femoris muscles is paralleled by a similar loss of strength of the external ocular muscles and that the loss of tonus of the external ocular muscles predisposes to forward dislocation of the eyeball.

The statistics make it clear that for large groups of patients the presence or absence of exophthalmos is correlated directly with the severity of hyperthyroidism, as measured by the basal metabolic rate and the presence of weakness of the quadriceps femoris muscles; also, that after 1930, when the severity of exophthalmic goiter was, on the whole, less than it was before 1922, the incidence of the association diminished significantly.

A great many authors have emphasized rapid fluctuation in the exophthalmos in individual patients,[31] and several of them have used this as an argument to support the theory that irritation of the cervical sympathetic nerves is the determining factor in the condition; they maintain that edema or some other gross anatomic alteration could not produce such rapid variations.[26] This, however, is incorrect. There is variation, but it is very slow and is not apparent from day to day, as has been frequently stated in both the old and the recent literature. The other ocular symptoms vary—especially the width of the palpebral fissure (Stellwag's sign), the retraction of the upper eyelids (Dalrymple's sign) and the lag of the eyelid (Graefe's sign) but measurements with the exophthalmometer taken frequently on the same patient indicate that any change in actual exophthalmos is insignificant, except as there may be a gradual increase or decrease extending over weeks, months or years.

The exophthalmos that accompanies the active stages of exophthalmic goiter is almost always moderate, as appears from a study of the tables. As a rule, it is bilateral and equal, although in many cases there is an appearance of asymmetry, which may or may not be due to a difference of 1 or 2 mm. of exophthalmos. Occasionally it is accompanied by weakness of the external ocular muscles, but strabismus is uncommon. It is accompanied by more or less marked spasm of the muscles of the eyelids, but there is seldom more than slight edema of the eyelids and conjunctivae, and rarely are there serious ocular complications, such as corneal ulcer and panophthalmitis. When exophthalmos is present its clinical course usually parallels the increase or decrease in the severity of the exophthalmic goiter; in a large majority of cases the eyes gradually return to their normal condition during the

31. Bulson, A. E., Jr., in discussion on Thayer, W. S.: Ocular Symptoms in Ophthalmic Goiter, Tr. Sect. Ophth., A. M. A. 1926, p. 56.

remissions which follow thyroidectomy or occur spontaneously. This is particularly true of the lower grades of exophthalmos. Exophthalmos is affected to a great extent by its previous duration and by its intensity. Some residual exophthalmos usually remains after a high degree of proptosis.

Exophthalmos in Exophthalmic Goiter with Low Basal Metabolic Rate.—The clinical data which have been presented were obtained from patients who had no previous operation on the thyroid gland, but the course of exophthalmos in cases of so-called recurrent exophthalmic goiter is no different. After a second resection of the thyroid gland exophthalmos, if present, usually recedes again, and although some degree of residual proptosis may remain in cases in which it was most marked, further progression is arrested.

However, there is a group of unusual cases in which exophthalmos first develops after thyroidectomy or, having been present before, increases afterward, even though the constitutional symptoms of the disease have abated and the basal metabolic rate has fallen to normal or even so far below normal as to provoke evidences of myxedema. In such cases resection of additional thyroid tissue is seldom of any benefit. Comparable to these cases there is a small number of cases in which exophthalmos develops after spontaneous remission from a previous elevation of the basal metabolic rate, and there is a still smaller number in which it may develop in the absence of any evidence of preceding hyperthyroidism. We have seen a few instances of each of these conditions. The clinical features which are common to all cases are a basal metabolic rate that is normal or lower than normal and a tendency to progress to a severe degree of proptosis, which is associated with marked edema of the eyelids. It is in this group that the vast majority of instances of so-called malignant exophthalmos develop.

Zimmerman [32] described 8 instances of the abnormality in patients who previously had undergone thyroidectomy. He called the condition "paradoxical exophthalmos." Three of the patients had had no ocular abnormalities; 3 had slight exophthalmos, and 2 others disclosed a Graefe sign at the time of the thyroidectomy. In all severe proptosis developed after the operation and was accompanied by edema of the eyelids, chemosis of the cornea and other more serious complications. In 3 the disturbance was limited to one eye; in the remainder it was bilateral. The basal metabolic rate in 3 was below — 14, and evidences of hypothyroidism were present in 4. Thompson [33] previously had reported 3 cases of acute edema of the orbit which developed after

32. Zimmerman, L. M.: Exophthalmos Following Operation for the Relief of Hyperthyroidism, Am. J. M. Sc. **178**:92 (July) 1929.

33. Thompson, E. S.: Orbital Edema in Exophthalmic Goitre, Am. J. Ophth. **7**:27 (Jan.) 1924.

thyroidectomy; in 1 case enucleation of both eyes was necessary. Zimmerman considered it possible that such edema may have been the factor responsible for the proptosis that was present in his cases. Two of the patients in Thompson's group had undergone thyroidectomy at the clinic, and our records disclose that the proptosis at the time of thyroidectomy was graded 3 + in one but only 1 + in the other.

Haines [34] described a case in which symptoms of exophthalmic goiter, including exophthalmos, were superimposed on those of myxedema which followed thyroidectomy. Burch [35] described a case in which a patient had progressive exophthalmos and a low basal metabolic rate, and Roeder [36] added 7 cases in which the patients had low basal metabolic rates; 3 of these patients had not undergone thyroidectomy previously. Roeder directed attention to the frequency of accompanying paralysis of the external ocular muscles. Interference with the function of the superior rectus muscles, which results in inability to elevate one or both eyeballs, is the usual abnormality, but we have secured photographs of 2 patients who had internal strabismus which was the result of paralysis of the external rectus muscles. King [37] added 5 cases in which patients who had exophthalmos also had low basal metabolic rates; Naffziger,[38] and Naffziger and Jones [39] added 6 similar cases.

Pathologic Observations.—It is from the specimens which have been obtained from orbital exploration, enucleation and decompression in cases of the type in question that most recent knowledge about the orbital content in the exophthalmos of exophthalmic goiter has been derived. This knowledge obviously applies only to this relatively infrequent and peculiar condition and may not be correctly informative as to the nature of the exophthalmos that occurs in the stages of exophthalmic goiter that are associated with elevated basal metabolic rates. The information that has been obtained from a study of the small group of patients who had malignant exophthalmos is as follows:

Benedict,[40] while exploring the orbits of 4 patients who had extreme proptosis, was impressed by the fact that all the tissues

34. Haines, S. F.: Exophthalmic Goiter and Myxedema: Report of a Case, Endocrinology **12**:55, 1928.

35. Burch, F. E.: The Exophthalmos of Graves' Disease, Minnesota Med. **12**: 668 (Nov.) 1929.

36. Roeder, C. A.: A Third Type of Toxic Thyroidism, Northwest Med. **29**: 395, 1930.

37. King, B. T.: The Cause of Exophthalmos, West. J. Surg. **39**:602 (Aug.) 1931.

38. Naffziger, H. C.: Pathologic Changes in the Orbit in Progressive Exophthalmos, Arch. Ophth. **9**:1 (Jan.) 1933.

39. Naffziger, H. C., and Jones, O. W., Jr.: The Surgical Treatment of Progressive Exophthalmos Following Thyroidectomy, J. A. M. A. **99**:638 (Aug. 20) 1932.

40. Benedict, W. L., cited by Pemberton.

were water-logged. There was no excessive fat in the orbit. The patients had basal metabolic rates that were normal or lower than normal. Two of these patients had undergone thyroidectomy. Another patient had not undergone thyroidectomy, although the diagnosis of exophthalmic goiter was definite; the basal metabolic rate on two determinations at the time of the enucleation of the eye was + 3 and + 11. Foster,[41] while dissecting "excessive fat" from the orbit of a patient who had exophthalmos, discovered that the muscles were greatly swollen. "Instead of being thin, flat, ribbon-like structures such as one becomes familiar with in squint operations, they had fusiform bellies not quite so stout as the last joint of one's little finger." The case of Burch, which has already been referred to, was that of a man aged 62. Three years previously, when he was examined at the clinic, he had a "stare" and a Graefe sign but no definite exophthalmos. The basal metabolic rate was determined twice; the first time it was + 20, and at another time it was + 30. The thyroid gland, which was removed, disclosed hypertrophied epithelium. Recovery from the thyroidectomy was complete; the patient remained well until 1928; then exophthalmos developed. Ultimately, both eyes had to be enucleated, and the muscles that surround and are attached to the orbit were found to average from 1 to 3 cm. in thickness. A cross-section disclosed muscle fibers which were in various stages of atrophy and were surrounded by loose fibrous tissue and edema. This seems to be the first detailed description of a peculiar pseudohypertrophy of the orbital muscles which, to judge from the subsequent reports of Naffziger and Naffziger and Jones, may act as a tumor in the production of exophthalmos. The complete reports of Naffziger and Naffziger and Jones deal with 6 cases in which Naffziger accomplished decompression of the orbit by his original operation. In this procedure the orbital plate of the frontal bone is resected. There was enormous hypertrophy of the rectus muscles in every instance. They were several times their normal thickness and were of unusual rubbery firmness. There were some interstitial edema and proliferation of lymphocytes. At necropsy in a similar case seen at the clinic, which is to be reported later, the orbits were disclosed to be literally packed with enlarged rectus muscles; this condition was similar to that described by Naffziger. There were also congestion of the vessels and considerable edema, which involved all the orbital tissues. The orbital fat was edematous, but was not excessive in amount.

A satisfactory explanation for this type of exophthalmos is difficult to discover. Mention has been made of the somewhat similar observations by Marine and Rosen, Friedgood and Kunde in experimental exophthalmos. The animals that had no thyroid glands and those that

41. Foster, M. R.: Exophthalmos and Limitation of the Eye Movements of Graves' Disease, Lancet **2**:701, 1920.

had low basal metabolic rates were those in which the most extreme grades of proptosis developed under the influence of the various agents employed. The possibility must be held in mind that some influence on the part of the pituitary body may cause man to be affected in a similar manner, but there is no clinical evidence to warrant such a conclusion. Another explanation is provided by the well known two product theory of H. S. Plummer. An abnormal product secreted by the diseased thyroid gland is responsible, according to this theory, for the exophthalmos in exophthalmic goiter but not for the elevation of the basal metabolic rate. The latter depends on the quantity of thyroxine, or normal product, which is supplied by the thyroid gland. The continued activity of the remnant of thyroid gland that remains after thyroidectomy might, under certain circumstances, supply enough of the abnormal product to provoke progression of the exophthalmos and yet not be able to supply enough thyroxine to maintain the basal metabolic rate. It would thus provoke exophthalmos in the presence of a basal metabolic rate no higher than those in cases of myxedema. Finally, there is some reason to believe that the peculiar edematous infiltration of the structures of the orbit which occurs with extreme degrees of exophthalmos and which develops in patients who have low basal metabolic rates is like the edematous infiltration which occurs infrequently as localized plaques of solid edema in the cutaneous and subcutaneous tissues of the head, trunk and extremities. The instances in which this has been observed are few, and in all of 8 cases reported by O'Leary [42] the patients had undergone thyroidectomy, which was followed by varying periods of remission of the symptoms of exophthalmic goiter. The improvement, in turn, was succeeded by the development of the plaques. The records of these cases bear a close resemblance to those of the cases of exophthalmos that are associated with low basal metabolic rates, and in some instances exophthalmos was an associated symptom. A similar process which involves the orbital structures may account for exophthalmos in exophthalmic goiter in a patient with a low basal metabolic rate.

When the investigator turns to the consideration of exophthalmos as it commonly occurs in association with both an elevated basal metabolic rate and the constitutional symptoms of hyperthyroidism, information is meager. Basedow, Möbius and other contributors to the older literature expressed the belief that an excessive amount of fat in the orbital cavity is an important factor, but this theory does not seem to have been supported. There is no positive evidence that excessive amounts of fat ever have been discovered in the orbital cavity. A feature of exophthalmic goiter is the disappearance

42. O'Leary, P. A.: Localized Solid Edema of the Extremities in Association with Exophthalmic Goiter, Arch. Dermat. & Syph. **21**:57 (Jan.) 1930.

of body fat, and it would be strange if fat persisted selectively in the orbit. It is possible that edematous infiltration of the orbital tissue has been mistaken for an increase in the amount of fat. A more plausible theory is the one which maintains that edema is responsible for protrusion of the eye, but this is objected to on the ground that accumulation of much fluid in the orbit would be accompanied by more swelling of the eyelids than ordinarily is seen. Mechanical theories, which depend principally on the assumption that engorgement of the vessels is necessary for the production of edema, have been advanced by King,[37] O'Day [43] and others.

It is difficult to discard the theories based on stimulation of the sympathetic nerves. The effect of sympathectomy in overcoming retraction of the eyelids in cases of exophthalmic goiter is undoubted, and in some cases proptosis also has been affected. The experiments of Labbé and his associates which relate to this question have been referred to.

There is a dearth of protocols of postmortem examinations bearing on the content of the orbits of patients who had exophthalmos and whose hyperthyroidism at the time of death was in the stage in which the basal metabolic rate is elevated and constitutional symptoms are present. As has been previously explained, the observations which recently have been made in cases in which the basal metabolic rate was low probably are not correctly informative as to what takes place in exophthalmic goiter when the basal metabolic rate is high. Our experience is no greater than that of others. Since the introduction of iodine as a routine measure in the treatment of exophthalmic goiter before thyroidectomy, the number of deaths has fallen to near the vanishing point, and permission to examine orbits in the few bodies that are available seems to be exceedingly difficult to obtain. What information we have was secured from Dr. L. B. Wilson,[44] who told us that in examinations which he made before 1917 of 8 patients who had active exophthalmic goiter with exophthalmos he found little fat, little edema and little venous congestion. The muscles were small and obviously degenerated, presenting an appearance exactly like that of the weak muscles of the thigh, which were studied in the same cases. Dr. Wilson expressed the tentative opinion that the exophthalmos could be explained by the weakness and relaxation of the rectus muscles. This conforms to the view which previously had been expressed by Traube, von Recklinghausen, Dalrymple and others, and it is supported by our finding of a positive correlation between weakness of the quadriceps extensor muscles and exophthalmos in the cases that have been reviewed for this article.

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43. O'Day, J. C.: Syphilis in the Rôle of Malignant Neoplasms, New York M. J. **100**:1258 (Dec. 26) 1914.

44. Wilson, L. B.: Personal communication to the authors.

SUMMARY

Forward displacement of the eyeball occurs in a variety of general diseases but is unusual except in exophthalmic goiter. In syphilis, tuberculosis, neurofibromatosis, multiple xanthomatosis and other conditions in which granulomatous masses or tumors may fill the orbit the explanation of any resulting exophthalmos is obvious. In the hemorrhagic diseases, including rickets, scurvy and the various forms of leukemia, intra-orbital hemorrhage may produce exophthalmos, which, under these circumstances, is perfectly understandable. In nephritis, hypertension and other conditions of so-called sympathicotonia, true proptosis is unusual, contrary to opinions recorded in the literature, although retraction of the eyelids and possibly other factors which contribute to a facial expression somewhat like that observed in exophthalmic goiter frequently are encountered.

The experimental production of exophthalmos must be evaluated with due consideration to the danger of applying to man observations that are made on animals, which normally are immune to exophthalmic goiter. Clinical experience indicates that the immediate course of exophthalmos in exophthalmic goiter is dependent on actual changes in the function of the thyroid gland.

In large groups of patients the presence or absence of exophthalmos in exophthalmic goiter is correlated directly with the severity of the hyperthyroidism, as measured by both the basal metabolic rate and the presence of weakness of the quadriceps femoris muscles. In the period after 1930, during which the severity of exophthalmic goiter has been milder, the incidence of associated exophthalmos has diminished to 40 per cent. Its incidence before 1922 was almost 70 per cent.

In exophthalmic goiter exophthalmos may develop or progress in two rather distinct phases of the disease: It may occur in association with an elevated basal metabolic rate and with the constitutional symptoms of the disease; it also may appear in an otherwise quiescent phase of the condition, when the basal metabolic rate is within, or even below, normal levels and when the constitutional symptoms are absent or at least attenuated. Exophthalmos which develops when the basal metabolic rate is elevated in exophthalmic goiter is usually moderate. Edema of the eyelids is present in only a small proportion of the cases and it is usually slight. The proptosis almost always is bilateral and is about equal or nearly equal in the two eyes; it usually is associated with spasm of the muscles of the lids and one or more of the characteristic signs which have been described by Dalrymple, Stellwag, Graefe, Möbius and W. H. Wilder. Weakness of the external ocular muscles, which is indicated by the occurrence of Möbius' sign, is common, but ophthalmoplegia and strabismus are unusual. Remission of the exophthalmos usually follows the decrease in the basal metabolic rate and

remission of the other manifestations of the disease which are brought about by thyroidectomy.

Exophthalmos which develops or progresses in the course of exophthalmic goiter when the basal metabolic rate is within, or even below, normal levels and when the constitutional symptoms are absent is unusual. Zimmerman referred to it as "paradoxical exophthalmos." Its degree, as a rule, is much greater than is that of the exophthalmos which is associated with an elevated basal metabolic rate. Edema of the eyelids and chemosis of the conjunctivae are the rule. They may be extreme and may lead to corneal ulceration and panophthalmitis, a condition which has been termed malignant exophthalmos. The exophthalmos in these cases is likely to be unilateral or to affect the eyes unequally. Ophthalmoplegia is common; on the other hand, spasm of the muscles of the eyelids is not marked, and in consequence the characteristic "stare" of exophthalmic goiter is little in evidence.

The exophthalmos which occurs with the elevated basal metabolic rate of exophthalmic goiter perhaps may be explained by spastic contraction of the orbital fibers of Müller's muscle, which acts against the weakened rectus muscles; the absence of mydriasis under these conditions may be accounted for by the observation of Labbé and his associates that thyroxine not only has a sympathomimetic action but also stimulates parasympathetic elements of the autonomic nervous system. Unfortunately, there are no recent reports of examination of the orbit in cases of this condition. The information which is available indicates that there is little fat, little edema and very little venous congestion and that the extra-ocular muscles are small and degenerated.

The explanation for the mechanism of the exophthalmos which occurs in patients who have low basal metabolic rates is not apparent. Why should the external rectus muscles become swollen and hardened and enlarge to several times their normal thickness? Why should edema develop in the eyelids when it was absent in the stage featured by a high basal metabolic rate? The evidence from the laboratory suggests that overfunction of the anterior lobe of the pituitary body may play a part in the production of this abnormality. Another possibility is presented by the two-product hypothesis of H. S. Plummer, which is based on the assumption that the abnormal product continues to act after the output of the normal product, thyroxine, has been curtailed. The edematous contents of the orbit are comparable in some respects to the localized subcutaneous areas of mucinous edema which are found in rare cases of exophthalmic goiter, particularly after thyroidectomy. Whatever the mechanism, it is difficult to understand why a few patients should show this paradoxical exophthalmos after subtotal resection of the thyroid gland for exophthalmic goiter when the large majority lose what exophthalmos they had before and show no sign of its recurrence. Further investigation of this question is under consideration.

ACUTE FOLLICULAR CONJUNCTIVITIS, BÉAL'S TYPE

A REPORT OF SEVEN CASES

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During the last two weeks of July 1934 five university students with acute follicular conjunctivitis were observed and treated at the University Hospital. The cases appeared to form a portion of a small epidemic, since on inquiry it was found that persons with a similar though milder condition were treated in the medical outpatient clinic. Two sporadic cases have also been seen in the past year. The clinical characteristics of the disease were (1) acute or subacute onset with much inflammation

Stereoscopic photograph of the left eye of A. J. M. five days after the onset of the symptoms. Note the massive follicular hypertrophy of the conjunctiva of the lower culdesac.

but little secretion, (2) mild nonpainful preauricular adenitis, (3) absence of inclusion bodies or pathogenic bacteria, (4) rapid evolution (from one to three weeks) and (5) serofibrinous secretion in which large mononuclear cells predominated.

The disease is typified by the following case:

A. J. M., aged 31, a graduate student, presented himself with the complaint of inflammation and secretion in the left eye of three days' duration. There was little discomfort. Examination revealed slight pseudoptosis of the affected eye and mild edema of the upper eyelid. The vision was 6/4.5 +. The cornea was clear on slit-lamp examination. The upper tarsal conjunctiva was hyperemic, infiltrated and granular. The conjunctiva of the lower fornix was thrown into folds which were studded with follicles, as shown in the illustration. There was marked

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hyperemia of the bulbar conjunctiva. The preauricular gland was enlarged but not tender. No pathogenic bacteria were seen in epithelial scrapings of smears of the secretion, nor were any recovered on culture. No inclusion bodies could be found on repeated examinations.

Four days after the onset of the disease in the left eye, the right eye became acutely involved. In thirty-six hours striking follicular hypertrophy developed which involved the entire conjunctiva but was most marked in the lower fornix. Bacteriologic examination of scrapings and secretion gave negative results and no inclusion bodies could be found in the epithelial cells. As in the left eye, the secretion was scanty and consisted mainly of mononuclear cells.

Treatment was begun with 1: 10,000 phenyl mercuric nitrate, and rapid healing of both eyes followed. At the end of three weeks only a few follicles persisted in the lower fornices. There was no secretion.

The clinical appearance of the eyes in these cases strongly suggested a diagnosis of adult inclusion conjunctivitis (swimming pool conjunctivitis). This possibility was eliminated, however, by the clinical course, which in the case of adult inclusion conjunctivitis is never less than two months. The absence of inclusion bodies also served to indicate that the infection was not caused by the virus of inclusion blennorrhea.

The five cases appeared to correspond, however, to the acute follicular conjunctivitis which was described by Béal [1] in 1907 and which has recently been discussed by Morax.[2] The latter described the disease under the name "acute follicular conjunctivitis, Béal's type."

Nothing is known concerning the causal agent of the condition. The absence of pathogenic bacteria suggests, however, that a virus is the etiologic factor.

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1. Béal, R.: Sur une forme particulière de conjonctivite aiguë avec follicules, Ann. d'ocul. **87**:1, 1907.

2. Morax, V.: Les conjonctivites folliculaires, Paris, Masson et Cie, 1933, p. 108.

DACRYADENITIS IN HYPERTHYROIDISM

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Orbital myositis in cases of exophthalmic goiter has been reported by Naffziger.[1] In all his cases thyroidectomy was followed by subjective relief, but in the course of several months there was progressive increase of exophthalmos. The basal metabolic rate following the operation was minus in each case. The extra-ocular muscles were seen to be from three to eight times their normal size and showed microscopically varying degrees of muscular degeneration, edematous fibrosis and cellular infiltration. Friedenwald [2] examined the extra-ocular muscles in six cases of hyperthyroidism in which the patient came to autopsy. In five of these cases the muscles were normal; in one case, however, in which exophthalmos had preceded by several months all other manifestations of hyperthyroidism, the extra-ocular muscles showed changes similar to those described by Naffziger. This case differed from those reported by Naffziger in that there was no operation followed by hypothyroidism. Friedenwald concluded that the changes in the orbital muscles are not a part of the usual picture of hyperthyroidism but that the condition is apparently a disease entity in which the lesion of the extra-ocular muscles is associated with abnormal functions of the thyroid but is not dependent on an increased or a decreased basal metabolism.

I have had the opportunity of examining the extra-ocular muscles of two [3] patients with progressive exophthalmos following thyroidectomy whose postoperative basal metabolic rate was minus. In each instance the changes in the muscles were identical with those described by Naffziger. It was noted, however, that the lesions may be limited to one or several of the muscles; therefore, from an examination of

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From the Department of Ophthalmology, Columbia University.

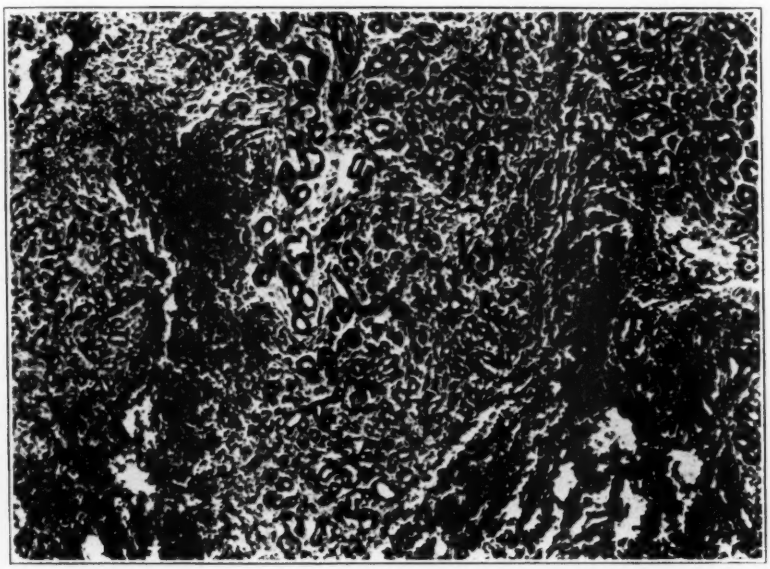
1. Naffziger, H. C.: Progressive Exophthalmos Following Thyroidectomy: Its Pathology and Treatment, Ann. Surg. **94**:582, 1931. Naffziger, H. C., and Jones, O. W., Jr.: The Surgical Treatment of Progressive Exophthalmos Following Thyroidectomy, J. A. M. A. **99**:638 (Aug. 20) 1932. McCool, J. L., and Naffziger, H. C.: The Pathological Changes in the Orbit in Progressive Exophthalmos, with Special Reference to Alterations in the Extra-Ocular Muscles and the Optic Discs, Tr. Am. Ophth. Soc. **30**:103, 1932.

2. Friedenwald, J. S.: Orbital Myositis and Choked Disc in Exophthalmic Goitre, Ann. Surg. **96**:995, 1932.

3. One was a patient of Dr. John M. Wheeler, and the other was a patient of Dr. R. N. Berke, admitted to Dr. John Dunnington's service.

one muscle or even of several muscles, it cannot be stated that these changes are not present in a given case.

The lacrimal glands in my cases were also examined, and lesions were noted similar to those in the muscles. The glandular tissue was in all stages of degeneration and was being replaced by edematous fibrous tissue, throughout which were numerous lymphocytes (figure). In places the lymphocytes were aggregated to form foci of considerable size. Histologically the lesion in the lacrimal gland was identical with those in the extra-ocular muscles. The orbital tissue intervening between the lacrimal gland and the extra-ocular muscles was apparently uninvolved. In other words, there appeared to be no diffuse orbital



Chronic dacryadenitis in a case of exophthalmic goiter.

inflammation, nor was there evidence that the inflammation had spread from one site to the other.

The observation of dacryadenitis in the cases just cited seems to explain the presence of the enlarged and easily palpable lacrimal glands noted by Naffziger in four of his six cases. Lacrimation was observed in all the cases, and in giving the usual clinical course he stated that after thyroidectomy "clinical improvement follows except that the exophthalmos does not disappear. In a variable period, often in three or four months, it becomes evident that the proptosis is increasing. As it proceeds, and increased fullness of the lids is noted; then lacrimation and epiphora appear."

It is not uncommon in a case of exophthalmic goiter to see a partial prolapse of the lacrimal gland when the patient looks down and the upper lid is pulled up. An enlargement of the gland may be at least partly responsible for the prolapse, and the enlargement itself may possibly be due to dacryadenitis which in turn causes lacrimation, a not infrequent symptom in cases of hyperthyroidism.

Clinical Notes

RETINOBLASTOMA: REPORT OF A CASE

A. Ray Irvine, M.D., Los Angeles

This case is reported on account of the rather unusual metastasis.

REPORT OF CASE

History.—A boy, aged 3½ years, came to me on Aug. 1, 1933. The parents had noticed a peculiar reflex in the pupil of the left eye. No redness, swelling or pain was noted. The child had a history of a fall two months prior to examination, at which time there was laceration of the skin above the left eye. There was no family history of malignant disease or trouble with the eyes.

Examination.—The right eye was normal. The left eye showed a dirty white mass in the vitreous which had the appearance of an exudate. Examination with the slit lamp showed the mass to be more or less firm and arranged in folds with a few small blood vessels over the surface. There was no perception of light. The tension of the eyeball was normal and the anterior chamber deep. A tentative diagnosis of tumor was made, and removal of the eye was advised. The parents objected to operative procedure.

The patient was not seen again until October 28. The mass had slightly increased in size and occupied most of the posterior chamber. At this time I was insistent on removal of the eye, but permission was again refused. I suggested consultation, and Dr. Weymann saw the patient and also advised operation.

The patient returned for the next visit on December 20. The tension was slightly increased. There was some discomfort, and the parents decided on operation. On December 21 enucleation was done.

Pathologic Appearance.—Macroscopic Description: The eye measured 2.4 cm. in the anterioposterior and 2.3 cm. in the lateral diameter. A segment of optic nerve measuring 2 mm. was attached. No deformity was noted externally, but immediately behind the lens a grayish-white cloud, which obscured the retina, could be observed.

Hemisection revealed the entire posterior chamber to be filled by a tumor to within 1 mm. of the lens. This mass seemed to be attached to the retina throughout its entire surface. The tumor was gray and cellular and flecked by yellow, gritty foci the size of a pinhead. It was extremely soft and friable.

Read at a meeting of the Western Ophthalmological Society, Butte, Mont., 1934.

From the Department of Ophthalmology of the University of Southern California.

About the entrance of the optic nerve the retina had been lifted away from the choroid, and a lenticular mass of tumor, 1.2 cm. in extent and from 2 to 3 mm. in thickness, had formed on each side of the papilla. Externally, the tumor was intimately adherent to the choroid, sclera and optic nerve.

Microscopic Description: The typical cell was of medium size, oval to cylindric and rather uniform throughout the lesion. The nuclei were large and conspicuous, oval or carrot-shaped and rather pale-staining except for one or

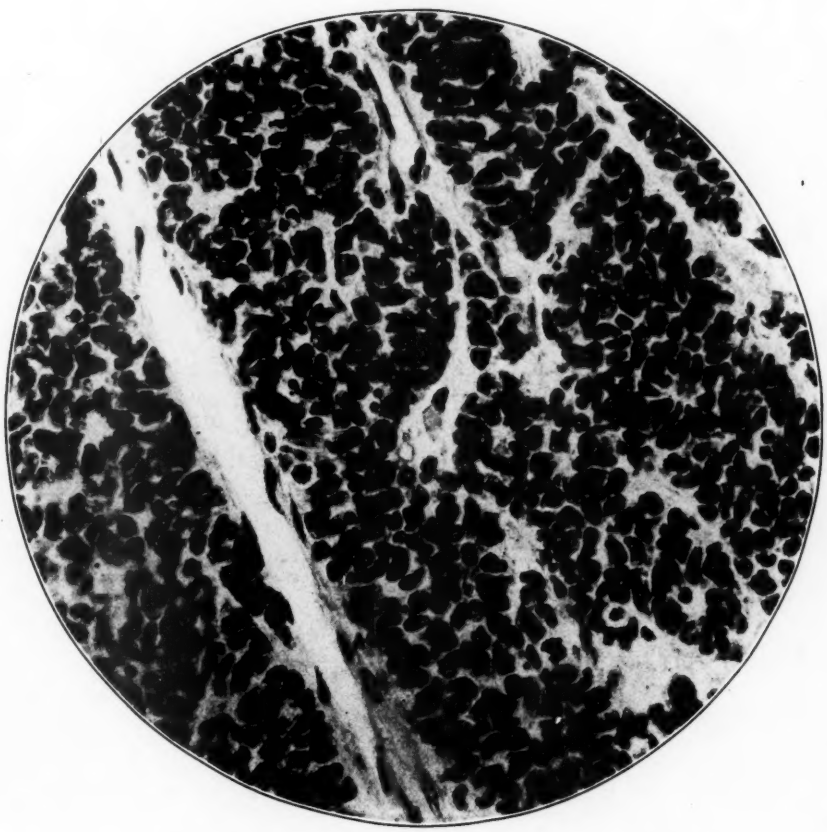


Fig. 1.—Section under a high power lens, showing tumor cells and tendency to rosette formation.

two small karyosomes. The cytoplasm seemed scanty and was frequently difficult to make out. No intercellular products were noted in the hematoxylin and eosin preparations.

The arrangement of the cells varied from place to place. At the anterior margin of the lesion they formed a mantle which extended over the surface of the retina. In the center of the posterior chamber perithelial growth was noted, the thin-walled, dilated vessels being surrounded by a cuff of viable tumor tissue

from eight to twenty cells thick. Necrotic tissue and inflammatory exudate occupied the remainder of the area. In places where the choroid had been invaded there was a tendency toward formation of large, lobulated, solidly packed masses of cells, although at the margin of infiltration columns and cords were noted. There was little tendency toward orderly arrangement, although in some of the larger cell masses definite alveoli or rosettes were seen.

Retrograde changes in the form of hemorrhage, necrosis and calcification were pronounced features, especially in the parts of the tumor removed from

Fig. 2.—Section under a low power lens, showing the tumor in the vitreous chamber, the invasion of the choroid, the separation of the retina and the invasion of the sclera.

the blood supply. Polymorphonuclear infiltration in the necrotic zones was abundant.

Invasion and destruction were also prominent. The retina except at its anterior extremity was completely destroyed, the choroid in its posterior half was diffusely infiltrated, the inner layers of the sclera were invaded and split

apart, and the optic nerve showed nests of tumor cells between the bundles of nerve fibers as far as the margin of resection.

Growth of the tumor was very active, as judged by the large number of mitoses present.

Diagnosis: The diagnosis was retinoblastoma of the eye.

Subsequent Course.— The patient made an uneventful recovery. On April 3, 1934, a small swelling was noted over the cheek bone on the side of the enucleation. The mass was rather firm and connected with the bone, and it quickly assumed the size of an ordinary walnut. The patient was referred to

Fig. 3.—Roentgenogram showing the shadow of the tumor seen from the floor of the left orbit.

St. Vincent's Hospital for irradiation under the supervision of Dr. Davis. The mass disappeared, but a second swelling appeared in two weeks in the frontal bone. A third mass, also in the frontal bone, appeared a week after the development of the first. Roentgenograms showed these masses to lie under the periosteum.

Dr. Orville N. Meland treated the patient with radium for a period of weeks, during which time a metastatic tumor appeared in the sternum. Several months later the patient died. An autopsy was not allowed.

The pathologic examination and report were made at St. Vincent's Hospital by Dr. J. W. Budd.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

THE AFFERENT PATH OF THE LIGHT REFLEX

A REVIEW OF THE LITERATURE

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AND

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Since the discovery of the Argyll Robertson or fixed pupil in 1869, many attempts have been made to explain the loss of the pupillary reactivity to light in this syndrome. In the majority of these the fixed pupil has been accounted for on the basis of a central interruption of the afferent path of the light reflex somewhere along its course to the oculomotor nucleus. A knowledge of the central course of the pathway of the light reflex has, therefore, assumed major importance for an explanation of the Argyll Robertson disorder, and in most of the publications on this subject either clinical or experimental evidence bearing on the localization of this pathway has been presented.

Extensive as this literature has been, it has not furnished satisfactory evidence concerning the course of that part of the arc situated within the interior of the brain, because this part of the arc has not until recently been accessible to experimental investigation. The use of a new and refined technic employing the Horsley-Clarke stereotaxic instrument has made it possible to fill this gap in our knowledge (Ranson and Magoun [1]) and to show that the path of the pupillary light reflex runs centrad from the optic tract and the superior quadrigeminal brachium to the pretectal region, or the transition area between the thalamus and the superior colliculus. From the pretectal region, the pathway swings ventrally around the rostral central gray matter of the aqueduct to the oculomotor nucleus, undergoing a partial crossing in the posterior commissure and ventral to the central gray matter. It

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This study was aided by a grant from the Rockefeller Foundation for research in neuro-anatomy.

1. Ranson, S. W., and Magoun, H. W.: The Central Path of the Pupillo-constrictor Reflex in Response to Light, Arch. Neurol. & Psychiat. **30**:1193 (Dec.) 1933.

may be emphasized that the pathway of the light reflex was found to be mediated by the pretectal region of the brain and not by the superior colliculus.

The essential rôle of the pretectal region in the innervation of the pupillary sphincter was next demonstrated in an investigation of the effect on the light reflex of small, accurately placed lesions made with the aid of the Horsley-Clarke instrument (Magoun and Ranson [2]). In this study it was found that if the pretectal region was almost completely destroyed on both sides of the brain, along with some damage to the rostromedial portion of the superior colliculus, the pupillary light reflex was completely and permanently abolished. Destruction of the superior colliculus alone was found to leave the light reflex unimpaired (Magoun [3]).

The following review represents an attempt to correlate these new observations with the results of other workers in this field. Although some of the literature which is covered has previously been considered by Adler,[4] the new point of view which has developed from recent researches justifies the reconsideration of this entire subject. The material at hand has been organized with reference to the portion of the brain stem under consideration, and we shall commence with the optic nerve and proceed centrally.

CORRELATION OF OBSERVATIONS

Optic Nerve.—The existence in the optic system of special elements subserving the activity of the light reflex has been debated in the literature. Gifford and Mayer [5] recently presented observations of their own and a summary of the clinical cases which have shown a marked difference in the functional activity of the retinal receptors for the light reflex and those for visual sensation. The presence of special pupilloconstrictor fibers in the optic nerve has been claimed on the basis of clinical material by Behr [6] and Abelsdorff.[7] The identity of these special elements is naturally difficult to establish on histologic grounds, but attempts have been made in this direction.

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2. Magoun, H. W., and Ranson, S. W.: The Central Path of the Light Reflex: A Study of the Effects of Lesions, Arch. Ophth., this issue, p. 791.

3. Magoun, H. W.: Maintenance of the Light Reflex After Destruction of the Superior Colliculus in the Cat, Am. J. Physiol. **111**:91 (Feb.) 1935.

4. Adler, F. H.: Pupilloscopic Findings in Lesions in Different Parts of the Reflex Arc, Arch. Ophth. **55**:262, 1926.

5. Gifford, S. R., and Mayer, L. L.: Retained Pupillary Reactions with No Perception of Light, Arch. Ophth. **6**:70 (July) 1931.

6. Behr, C.: Zur Physiologie und Pathologie des Lichtreflexes der Pupille, Arch. f. Ophth. **86**:468, 1913.

7. Abelsdorff, G.: Zur Frage der Existenz gesonderter Pupillarfasern im Sehnerven, Klin. Monatsbl. f. Augenh. **62**:170, 1919.

Gudden [8] examined the atrophy in the optic nerve of a rabbit which had been made blind, with retention of the light reflex, by a lesion of the superior colliculus. Most of the atrophy in the optic nerve was confined to the small fibers, and these, he concluded, effected the visual activity, which had been lost. The fibers remaining in the optic nerve were mainly large, and these large fibers, Gudden suggested, mediated the light reflex, which had been retained.

More recently Ingvar [9] ascribed the activity of the light reflex to fibers of the opposite extreme in size, to unmyelinated optic fibers. Claiming the presence of unmyelinated fibers in the optic nerve of the rabbit, cat, monkey and man, Ingvar reported that these fibers assumed a peripheral concentration at the chiasma and the adjacent portion of the optic tract, and in clinical syphilitic material in which there had been a loss of the light reflex he found a destruction of the periphery of the optic chiasma. It is difficult to reconcile the peripheral situation of the path of the light reflex, claimed by Ingvar with the central position in the optic chiasma and tract of degeneration from the macula (Brouwer and Zeeman [10]), the lighting of which yields the best pupilloconstriction of any part of the retina (Hess [11]). Moreover, the recent work of Arey and Schaible [12] shows Ingvar's claim of unmyelinated fibers in the optic nerve to be without support. In this study, Arey and Schaible made fiber counts in material stained with silver and osmic acid and found no evidence of the existence of appreciable numbers of unmyelinated fibers in the optic nerve of the rabbit, dog, monkey and human being.

The question of the separate identity of the elements of the light reflex in the optic system has therefore hardly been more than proposed. The presence of any special pupillomotor fibers is denied by Hess.[11] It seems clear, however, from a study of the relationship of the path of the light reflex to the optic chiasma, which will be discussed immediately, that only a portion of the optic fibers mediate the light reflex; whether they do so by virtue of special connections or because of some intrinsic difference from the remainder of the optic fibers is

8. von Gudden: Demonstration der Sehfasern und Pupillarfasern des Nervus opticus, Sitzungsb. d. Gesellsch. f. Morphol. u. Physiol. **1**:169, 1886.

9. Ingvar, S.: On the Pathogenesis of the Argyll-Robertson Phenomenon, Bull. Johns Hopkins Hosp. **43**:363, 1928.

10. Brouwer, B., and Zeeman, W. P. C.: The Projection of the Retina in the Primary Optic Neuron in Monkeys, Brain **49**:1, 1926.

11. Hess, C. (posthumously completed by G. Groethuysen): "Pupille," in Bethe, A.; von Bergmann, G.; Embden, G., and Ellinger, A.: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1929, vol. 12, p. 176.

12. Arey, L. B., and Schaible, A. J.: The Nerve-Fiber Composition of the Optic Nerve, Anat. Rec. **58**:3, 1934.

unknown. When the term "pupillomotor or afferent light reflex fiber" is employed in the following discussion it is used for convenience only, with no connotation of histologic specialty.

Optic Chiasma.—The decussation of the pathway of the light reflex in the optic chiasma follows in a general way the fundamental plan of crossing of optic fibers there. Quantitative differences in some animals in the amount of crossing of the pupillomotor fibers and the optic fibers of other functions do exist, however. In the mammalian transition from a total to a partial crossing in the chiasma, the number of uncrossed pupillomotor fibers appears to lag behind the number of uncrossed optic fibers of other function in both rodents and carnivores. The presence of these uncrossed pupillomotor fibers in the optic chiasma may be detected by lighting one eye after section of the contralateral optic tract. Any reaction to light obtained under these conditions must be mediated by fibers which remained uncrossed at the optic chiasma.

According to Knoll,[13] cutting one optic tract in the rabbit totally abolishes the light reflex from the contralateral eye. If this observation is correct, it can mean only that the light reflex fibers are totally crossed in the optic chiasma of the rabbit. Yet a series of anatomic studies shows the presence of uncrossed optic fibers in the chiasma of the rabbit, and it thus appears that there are a number of optic fibers in the rabbit which do not mediate the light reaction.

A comparable situation is found in the cat. After cutting one optic tract in the cat, Karplus and Kreidl [14] obtained only a trace of constriction from lighting the contralateral eye. Our own results [2] show that only a trace of reaction is obtained from lighting the contralateral eye following a lesion of one half of the pretectal region in the cat. While these observations demonstrate the presence of uncrossed pupillomotor fibers in the chiasma of the cat, they show that these fibers are few in number, since they can occasion only a trace of pupilloconstriction. Anatomic studies, however, reveal the presence of a large number of uncrossed optic fibers in the chiasma of the cat (Barris, Ingram and Ranson [15]). This excess of optic fibers of indeterminate function over the component which can be demonstrated to mediate the light reflex apparently indicates that in the cat, as in the rabbit, not all of the optic fibers serve to mediate the light reflex.

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13. Knoll, P.: Beiträge zur Physiologie der Vierhügel, Beitr. z. Anat. u. Physiol. **4**:109, 1869.

14. Karplus, J. P., and Kreidl, A.: Ueber die Bahn des Pupillarreflexes (Die reflektorische Pupillenstarre), Arch. f. d. ges. Physiol. **149**:115, 1912.

15. Barris, R. W.; Ingram, W. R., and Ranson, S. W.: Optic Connections of the Diencephalon and Midbrain of the Cat, J. Comp. Neurol., to be published.

In primates the numbers of crossed and uncrossed pupillomotor fibers in the optic chiasma appear to be about equal. After cutting one optic tract in the monkey, Bernheimer [16] observed little difference in the reactions of the two pupils to light, and no difference was noted by Karplus and Kreidl.[14] The latter authors specifically stated that the reactions were not hemianopic. In man the light reaction following a lesion of one optic tract has been recognized as hemianopic and has become known as Wernicke's reaction.

Optic Tract.—One of the few uncontroverted points regarding the path of the light reflex is its central passage in the optic tract. The stretch of optic tract over the hypothalamus from which Karplus and Kreidl [14] obtained dilatation of the pupil on electrical stimulation was not encountered by us,[1] and it is probable that their explanation of the phenomenon, as being due to a spread of current to the pupillodilator region of the hypothalamus, is correct.

Posterior Accessory Optic Tract.—The possibility that light reflex fibers are contained in the posterior accessory optic tract has been considered by a number of workers since Perlia,[17] in material stained by the Marchi method, traced optic degeneration in the rabbit by way of this tract to the anterior portion of the oculomotor nucleus. But the uncertainty of the occurrence of this tract in the higher mammals was pointed out by Marburg [18] in opposition to the idea of its mediating the light reflex, and the work of Barris, Ingram and Ranson [15] indicates that it may be an occipitopontile rather than a primary optic path in the cat. Our stimulation of its region of transit in the cat [1] never yielded any constrictor responses. Moreover, the abolition of the light reflex by lesions of the brachium of the superior colliculus (Karplus and Kreidl [14]) and by lesions of the pretectal region,[2] which leave intact any possible connections of the posterior accessory optic tract with the oculomotor nucleus, certainly demonstrated that this tract contains no afferent light reflex fibers.

Lateral Geniculate Body.—A consideration of the relationship of the path of the light reflex to the lateral geniculate body may be limited to its dorsal nucleus, since the ventral nucleus is a structure which becomes vestigial in primates (Le Gros Clark [19]) and since in the cat it probably does not receive any optic fibers (Barris, Ingram and Ranson [15]).

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16. Bernheimer, S.: Die Reflexbahn der Pupillarreaction, Arch. f. Ophth. **47**:1, 1898.

17. Perlia: Die Anatomie des Oculomotoriuscentrums beim Menschen (Nachtrag), Arch. f. Ophth. **35**:287, 1889.

18. Marburg, O.: Basale Opticuswurzel und Tractus peduncularis transversus, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **10**:66 1903.

19. Clark, W. E. L.: A Morphological Study of the Lateral Geniculate Body, Brit. J. Ophth. **16**:264, 1932.

It has been mentioned that lighting the macula produces the best pupilloconstriction of any part of the retina (Hess [11]). Yet Brouwer and Zeeman [10] were unable in material stained by the Marchi method to trace macular degeneration centrad of the lateral geniculate body in the monkey. If this observation is correct, it apparently admits of two possibilities: Either a synapse occurs in the path of the light reflex at the lateral geniculate body, or this pathway is unmyelinated, at least from the lateral geniculate body centrad.

That the pupillomotor fibers are unmyelinated has been suggested by Ingvar.[9] Lenz also has been quoted (Duke-Elder [20]) in support of this. Lenz,[21] in human material stained by the Bielschowsky method, was able to trace a fascicle of optic fibers from the region of the lateral geniculate body to the pretectal region and thence to the oculomotor nucleus. We have not been able to find any definite statement in Lenz' paper, however, as to whether these fibers were unmyelinated or not. The results of Arey and Schaible,[12] who found no evidence of the existence of appreciable numbers of unmyelinated fibers in the optic nerve, and the observations of a series of workers who have traced optic fibers into the pretectal region, using the Weigert or Marchi stain, seem to indicate that the weight of evidence is opposed to the conception that the path of the light reflex is made up of unmyelinated optic fibers. The myelinated optic connections with the pretectal region will be discussed in detail later.

The possibility of a synapse in the path of the light reflex at the lateral geniculate body is contrary to all of the available evidence. First, the dorsal nucleus of the lateral geniculate body appears to stand in exclusive relationship to the striate area of the cerebral cortex. This is apparent on broad comparative neurologic grounds. In addition, after a lesion of the striate area in the monkey, complete degeneration of all the cells of the dorsal nucleus of the lateral geniculate body was found by Poliak.[22] This was shown in another way by Barris, Ingram and Ranson,[15] who found only geniculocortical degeneration in the cat after a lesion of the dorsal nucleus of the lateral geniculate body. Secondly, Karplus and Kreidl [14] obtained no constriction of the pupil from electrical stimulation of the lateral geniculate body, and they came to the conclusion that the path of the light reflex ran centrad ventral to that structure. In our stimulation of the region of the lateral geniculate body in the cat,[1] a few marked and many weak constrictor reactions

20. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, vol. 1.

21. Lenz, G.: Untersuchungen über die intrazerebrale Bahn des Pupillarreflexes, Ber. u. d. Versamml. d. deutsch. ophth. Gesellsch. **46**:140, 1927.

22. Poliak, S.: The Main Afferent Fiber System of the Cerebral Cortex in Primates, Berkeley, Calif., University of California Press, 1932, vol. 2.

were obtained from the stimulation of points near the dorsal surface of the optic tract in the lateral geniculate body. But since these results were also obtained from points near the ventral surface of the optic tract in the medial geniculate body, we have interpreted them as being due to spread of current to the fibers of the adjacent optic tract. A third point in opposition to the possibility of a synapse in the path of the light reflex at the lateral geniculate body is the fact that the light reflex was found to remain intact after a more or less complete destruction of the lateral geniculate body in the dog by Bechterew [23] and in several clinical cases by Henschen.[24]

Brachium of the Superior Colliculus.—In stimulation experiments on the cat and monkey, Karplus and Kreidl [14] were able to follow the path of the light reflex centrad over the optic tract into the brachium of the superior colliculus and along the furrow at the anterior border of the superior colliculus almost to the midline. They found that section of the brachium of the superior colliculus abolished the light reflex.

Spiegel and Nagasaka [25] observed, however, that a lesion of the medial portion of the superior colliculus in the rabbit, cat and dog left the light reflex intact and so suggested that only a portion of the pupillomotor fibers continued mediad of the brachium, while the remainder, large enough to mediate the light reflex, made connection with the oculomotor nucleus through the lateral portion of the superior colliculus.

Our results in the cat [1] demonstrate that no constrictor response can be obtained from the stimulation of points in the superior colliculus and that from the superior quadrigeminal brachium points yielding pupilloconstriction on stimulation may be followed medially and rostrally into the pretectal region. Furthermore, we have produced complete abolition of the light reflex in cats by bilateral lesions of the pretectal region and rostromedial portion of the superior colliculus, which have left the brachium and the lateral portion of the superior colliculus intact.[2] Destruction of the superior colliculus alone was found to leave the light reflex unimpaired,[3] and the evidence taken together clearly demonstrates that the light reactions are effected exclusively through the pretectal region.

It has been generally assumed, however, that the pupilloconstrictor impulses from the superior quardigeminal brachium enter the superior

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23. Bechterew, W.: Ueber den Verlauf der die Pupille verengenden Nervenfasern im Gehirn und über die Localisation eines Centrums für die Iris und Contraction der Augenmuskeln, Arch. f. d. ges. Physiol. **31**:60, 1883.

24. Henschen, S. E.: Klinische und anatomische Beiträge zur Pathologie des Gehirns, Upsala, Almquist & Wiksell, 1896, pt. 3.

25. Spiegel, E. A., and Nagasaka, G.: Experimental Studien am Nervensystem: VI. Ueber die Beziehung des Pupillenreflexbogens zum vordern Vierhügel, Arch. f. d. ges. Physiol. **215**:120, 1927.

colliculus and thence reach the oculomotor nucleus by way of the tectobulbar fibers. Before we proceed to a discussion of the pretectal region, therefore, the large amount of negative evidence relating to the superior colliculus may be considered.

Superior Colliculus.—It has just been mentioned that by stimulating the dorsal surface of the brain stem in the cat and monkey, Karplus and Kreidl [14] were able to follow pupilloconstrictor responses mediad from the brachium along the furrow at the anterior border of the superior colliculus. But when these workers stimulated the superior colliculus itself, dilatation of the pupil ensued. The same result had been obtained previously in the rabbit by Knoll [13] and in the rabbit, cat, jackal, dog and monkey by Ferrier.[26] Our results [1] showed that with a localized stimulus no response was obtained from the pupil until the electrode passed through the superior colliculus and reached the underlying central gray matter of the aqueduct or the tegmentum of the midbrain, where stimulation caused pupillodilatation. The fact is clear that stimulation of the superior colliculus does not produce constriction of the pupil.

Furthermore, the light reflex has been found to remain intact after destruction of the superior colliculus by Knoll [13] in the rabbit, Bechterew [23] in the dog and pigeon (corpora bigemina), Gudden [8] in the rabbit, Ferrier and Turner [27] in the monkey, Levinsohn in the rabbit [28] and in the monkey,[29] Bernheimer [30] in the monkey and Keller and Stewart [31] and ourselves [3] in the cat.

Some of this work has been justly criticized by Wilson [32] on the grounds of the possibility of incomplete lesions through lack of histologic verification of the extent of destruction. This criticism cannot be applied to the more recent investigations, however, and the unity of result is striking and uncontradicted. Wilson made the interesting suggestion of a rostrocaudal localization of function within the tectofugal systems, the most rostral of these serving to mediate the light reflex. This suggestion may be brought into line with our results and

26. Ferrier, D.: The Functions of the Brain, ed. 2, London, Smith, Elder & Co., 1886.

27. Ferrier, D., and Turner, W. A.: Experimental Lesions of the Corpora Quadrigemina in Monkeys, Brain **24**:27, 1901.

28. Levinsohn, G.: Beiträge zur Physiologie des Pupillarreflexes, Arch. f. Ophth. **59**:191 and 436, 1904.

29. Levinsohn, G.: Experimental Untersuchungen über die Beziehungen des vorderen Vierhügels zum Pupillarreflex, Arch. f. Ophth. **72**:367, 1909.

30. Bernheimer, S.: Weitere experimentelle Studien zur Kenntnis der Lage des Sphinkter- und Levator Kerns, Arch. f. Ophth. **70**:539, 1909.

31. Keller, A. D., and Stewart, L.: The Superior Colliculi and the Pupillary Light Reflex in the Cat, Am. J. Physiol. **101**:64, 1932.

32. Wilson, S. A. K.: Some Problems in Neurology: I. The Argyll Robertson Pupil, J. Neurol. & Psychopath. **2**:1, 1921.

with those of others just mentioned if the schema is simply shifted farther rostrally, so that pupillomotor activity may be effected through the pretectal region.

Pretectal Region.—The anatomic entity of the pretectal region, or transition area between the thalamus and the tectum, has only recently come to be widely recognized, largely perhaps through the investigations of Le Gros Clark.[33] For this reason there is much investigative material bearing on it which does not refer to the region by name. This material includes results from electrical stimulation, study of the effect on the light reflex of pretectal lesions and anatomic investigations on the structure and connections of this region.

It may be recalled once more that Karplus and Kreidl [14] were able to trace constrictor responses in the cat and monkey from the brachium of the superior colliculus toward the midline in the furrow at the anterolateral border of the superior colliculus. Somewhere between the brachium and the midline the pupillomotor fibers sank below the surface of the brain stem and became lost to further surface stimulation.

By using the Horsley-Clarke apparatus we have systematically stimulated this region in the cat,[1] corroborating the findings of Karplus and Kreidl [14] as far as they went and tracing the remainder of the course of the pupillomotor pathway. With this method of stimulation we have found that after leaving the brachium of the superior colliculus the pupillomotor fibers turn rostromedially and enter the pretectal region. After a partial decussation in the posterior commissure they arch ventrally around the rostral end of the central gray matter of the aqueduct, with a partial ventral decussation, to reach the rostral portion of the oculomotor nucleus. The evidence bearing on these decussations will be discussed later.

Chance observations of constriction of the pupils from stimulation of the pretectal region have been made before. Knoll [13] observed a marked pupilloconstriction from stimulation in the rabbit when the electrodes were placed on either side of the vena magna Galeni "where both optic nerves run in contact with one another." Sachs [34] noted a constriction of the pupils in the monkey from stimulation at the point "where the outer part of the posterior commissure crosses behind Meynert's bundle."

An impairment of the reactions of the pupils to light has been noted following lesions of the pretectal region by some of the very workers who have observed that the light reflex remained intact following corresponding lesions of the superior colliculus. Gudden [8] found that while removal of the superior colliculus in the rabbit did not impair pupil-

33. Clark, W. E. L.: The Structure and Connections of the Thalamus, Brain **55**:406, 1932.

34. Sachs, E.: On the Structure and Functional Relations of the Optic Thalamus, Brain **32**:95, 1909.

lary movement, if one performed the same experiment "farther forward in the thalamus" the pupils became extraordinarily dilated. Bechterew [23] observed that although destruction of the superior colliculus in the dog left the pupillary reactions intact, a lesion in the "posterior part of the thalamus" led to reduction of the light reflex.[35] Bernheimer [16] was able to show that when the superior colliculus of the monkey was completely removed without injury to the bordering brain parts against the thalamus, the pupillary reflex remained unimpaired. But when the injury included "a part of the Sulcus thalami," a slight widening and a slower reaction of the pupil could be observed for several days.

The inaccessible position of the pretectal region, covered dorsally and laterally by the cerebral hemisphere and cut off from a ventral approach by the depth of the brain stem, has doubtless prevented further experimental destruction of this nature. By using the Horsley-Clarke apparatus, however, we have been able to introduce an accurately oriented electrode through the indifferent cerebral cortex and to produce either bilateral or unilateral electrolytic lesions of the pretectal region in a large number of cats.

A study of the light reactions after the production of such lesions [2] and after the production of lesions of the superior colliculus [3] has demonstrated that the pupillary light reflex is mediated exclusively by the pretectal region and that no part of the superior colliculus is concerned with this reaction.

The observations both from electrical stimulation and from the study of the effects of lesions demonstrate the importance of the pretectal region in the pathway of the light reflex. The anatomic evidence bearing on the connection of the optic tract with this region should, therefore, be examined in order to determine what structural basis is at hand to account for this evidence obtained by experimental procedures.

Lenz,[21] working with human material stained by the Bielschowsky method, traced a bundle of optic fibers through the pretectal region to the posterior commissure. Myelinated fibers from the optic nerve have been traced into the pretectal region by a number of workers. Darkshevich [36] traced such a fascicle of primary optic fibers in the normal rabbit and dog in material stained by the Weigert method. He designated this fascicle as the path of light reflex, but unfortunately he thought that it terminated in the habenula and the pineal gland. Probst [37] followed this pretectal fascicle in the rabbit in material stained

35. Bechterew, W.: Die Leitungsbahnen im Gehirn und Rückenmark, Leipzig, A. Georgi, 1899.

36. Darkshevich, L.: Ueber die sogenannten primären Opticuscentren und ihre Beziehung zur Grosshirnrinde, Arch. f. Anat. u. Physiol. (Anat. Abt.) **1**:249, 1886.

37. Probst, M.: Ueber den Verlauf der Sehnervenfasern und deren Endigung im Zwischen- und Mittelhirn, Monatschr. f. Psychiat. u. Neurol. **8**:165, 1900.

by the Marchi method after enucleation of the eye and pointed out that it did not enter the habenula. Degeneration in the pretectal region after long-standing enucleation of the eye was recognized by Minkowski [38] in the rabbit, goat and cat. A pretectal fascicle was identified by Le Gros Clark [39] in the rat in material showing optic degeneration stained by the Marchi method. The course and distribution in the pretectal region of these myelinated fibers have been carefully worked out with the Marchi method following optic degeneration in the cat by Barris, Ingram and Ranson.[15] There is, therefore, abundant evidence for the existence of myelinated optic connections with the pretectal region.

Following pretectal lesions in the rat, Papez and Freeman [40] were able to trace degenerated fibers across the posterior commissure and down to the ciliary nucleus of the opposite side, the Marchi staining method being used. The fact that optic degeneration has been traced to the pretectal region but not across the posterior commissure by Barris, Ingram and Ranson, Le Gros Clark and Probst, and that degeneration from the pretectal region has been followed across the posterior commissure and down to the sphincter nucleus of the oculomotor nerve by Papez and Freeman, suggests the possibility that a synapse in the pathway of the light reflex may occur in the pretectal region.

Posterior Commissure.—It has long been recognized that in addition to its crossing in the optic chiasma a central decussation of the pathway of the light reflex must occur. In rodents and the vertebrates below them, in which all of the pupillomotor fibers cross to the opposite side of the brain in the optic chiasma, only a direct light reaction is obtained from lighting one retina. The necessity of a second total crossing, centrally, in order to bring these impulses back to the ipsilateral side to produce the direct reaction was pointed out as long ago as 1890 by Steinach.[41]

In higher mammals a partial central decussation of the light reflex fibers which remain uncrossed in the optic chiasma also takes place. After midsagittal section of the optic chiasma any consensual reaction obtained from lighting one retina must be mediated by impulses crossing to the opposite side centrally, and a consensual as well as a direct

38. Minkowski, M.: Ueber den Verlauf, die Endigung und die zentrale Repräsentation von gekränzten und ungekränzten Sehnervenfasern bei einigen Säugetieren und beim Menschen, Schweiz. Arch. f. Neurol. u. Psychiat. **6**:201, 1920; **7**:268, 1920.

39. Clark, W. E. L.: Degeneration of Optic Tract Fibers in the Rat, J. Anat. **66**:138, 1931.

40. Papez, J. W., and Freeman, G. L.: Superior Colliculi and Their Fiber Connections in the Rat, J. Comp. Neurol. **51**:409, 1930.

41. Steinach, E.: Untersuchungen zur vergleichenden Physiologie der Iris, Arch. f. d. ges. Physiol. **47**:289, 1890.

reaction was obtained from lighting either eye after midsagittal section of the optic chiasma in the dog by Bechterew [35] and in the monkey by Bernheimer.[16]

It is natural to look for the situation of this central decussation in that part of the brain where the light reflex fibers approach the midline. The close association of the pretectal region and the posterior commissure calls attention to the latter structure, and the evidence bearing on a crossing of the pathway of the light reflex in the posterior commissure may now be presented.

Darkshevich [42] found a reduction of the light reaction after section of the posterior commissure in the rabbit, and he concluded that this commissure formed a part of the path of the light reflex. Following section of the posterior commissure in the cat, the light reactions were noted by Harris [43] to suffer a transitory impairment and then become normal. Karplus and Kreidl [14] after section of the posterior commissure in the cat obtained a bilateral pupilloconstriction from stimulation of one brachium of the superior colliculus, and both Harris and Karplus and Kreidl considered that their results disproved the contention of Darkshevich [42] that the pupilloconstrictor pathway crossed in the posterior commissure. In examination of the autopsy material from a patient who had exhibited loss of the light reflex, Collier [44] found a hemorrhagic softening on one side of the posterior commissure, and in another such patient Herrmann [45] found a plaque which destroyed the posterior commissure but which did not extend beyond it. Recently, Spiegel [46] found a reduction in the light reflex following section of the posterior commissure in the cat.

In our stimulation experiments on this region of the cat's brain,[1] a bilateral constriction, of an equal amount in the two pupils, was obtained from stimulation both of the posterior commissure and of the pretectal region dorsolateral to it. Excitation of the fibers surrounding the rostral central gray matter of the aqueduct, between the posterior commissure and the oculomotor nucleus, produced, however, a marked

42. Darkshevich, L.: Versuche über die Durchschneidung der hinteren Gehirncommissur beim Kaninchen, Arch. f. d. ges. Physiol. **38**:120, 1886.

43. Harris, W.: Binocular and Stereoscopic Vision in Man and Other Vertebrates, with Its Relation to the Decussation of the Optic Nerves, the Ocular Movements and the Pupil Light Reflex, Brain **27**:107, 1904.

44. Collier, J.: Nuclear Ophthalmoplegia, with Especial Reference to Retraction of the Lids and Ptosis and to Lesions of the Posterior Commissure, Brain **50**:488, 1927.

45. Herrmann, G.: Zur Frage der morphologischen Beziehungen des Argyll-Robertson, Allg. Ztschr. f. Psychiat. **88**:353, 1928.

46. Spiegel, E. A.: Further Experiments on the Localization of the Argyll Robertson Phenomenon (Injuries to the Posterior Commissure), in Volume jubilaire en l'honneur du Profeseur G. Marinesco, Bucharest, Société Roumaine de Neurologie, Psychiatrie et Endocrinologie, 1933, p. 625.

ipsilateral and a weaker contralateral constriction. The apparent explanation of these facts is that there is a partial crossing of the path of the light reflex both in the posterior commissure and ventral to the central gray matter.

The decussation of the light reflex fibers in the posterior commissure has been investigated further by studying the effect on the light reflex of midsagittal lesions of that commissure in the cat (Magoun, Ranson and Mayer [47]). After destruction of the posterior commissure in the midline, the light reactions of both eyes were found to be reduced but not abolished, indicating, as did the results from the stimulation experiments, that there is a partial but not a complete central decussation of the pathway of the light reflex in the posterior commissure.

Fibers Surrounding the Central Gray Matter of the Aqueduct.—In our stimulation experiments on the cat,[1] pupilloconstrictor responses were followed from the pretectal region and the posterior commissure toward the oculomotor nucleus by way of the fibers arching ventrocaudally around the rostral end of the central gray matter of the aqueduct. It is noteworthy that this is the identical situation of the degeneration from pretectal lesions found by Papez and Freeman [40] in the rat. A marked ipsilateral and a weaker contralateral pupilloconstriction from stimulation of these fibers indicate that, while the larger part of the light reflex impulses which they mediate reach the oculomotor nucleus of the same side, some cross to the opposite side of the brain ventral to the central gray matter.

The problems concerning the portion of the oculomotor nucleus which innervates the pupillary sphincter through the ciliary ganglion are so many as to form a special topic, consideration of which is beyond the scope of this review.

CONCLUSION

It is clear from a survey of the evidence which has been presented that enough data are at hand to permit a reconstruction of the afferent course of the pathway of the pupillary light reflex. This pathway runs centrad in the optic tract and the superior quadrigeminal brachium to the pretectal region. From the pretectal region it swings ventrally around the central gray matter enclosing the rostral end of the cerebral aqueduct to reach the oculomotor nucleus. The pathway undergoes a partial crossing in the posterior commissure and another crossing somewhere ventral to the central gray matter. The question of whether special elements in the optic system subserve the activity of the light reflex is still uncertain. The evidence is clear that no part of the superior colliculus is concerned with the pupillary light reflex.

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47. Magoun, H. W.; Ranson, S. W., and Mayer, L. L.: The Pupillary Light Reflex After Destruction of the Posterior Commissure in the Cat, Am. J. Ophth., to be published.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

SOCIETY NOTES

International Association for Prevention of Blindness.—Vice President Dr. Park Lewis of Buffalo presided over the annual conference of the International Association for the Prevention of Blindness, in the absence of President de Lapersonne. Delegates from most of the thirty-four nations represented in the association attended the conference, which was held in London on April 5. Dr. A. Fraceschetti of Geneva, Switzerland, advocated among other measures the special training of physicians, particularly ophthalmologists, in genetics; making available facilities for the sterilization of men and women afflicted with hereditary diseases of the eye, and a decrease in consanguineous marriages. Dr. M. Van Duyse of Ghent, Belgium, cited the fact that the chances of becoming blind are greatest in the first two years of life, owing chiefly to ophthalmia neonatorum and corneal lesions. The American delegate, Mr. Lewis H. Carris of New York, stated that during the past twenty-five years the United States has achieved a 75 per cent reduction in the number of cases of blindness from ophthalmia neonatorum among children entering schools for the blind.

New York State Medical Society.—The Section on Ophthalmology of the New York State Medical Society will hold its meeting on May 14 in the senate chamber of the state capitol, Albany. The following program will be given: "A Study of the Pathology in Cases of Sympathetic Ophthalmia," Dr. Bernard Samuels, New York; "Treatment of Sympathetic Ophthalmia with Antidiphtheric Serum," Dr. Frederick H. Verhoeff, Boston (discussion opened by Dr. Ben Witt Key, New York); "Allergy in Its Relation to Sympathetic Ophthalmia," Dr. Alan C. Woods, Baltimore (discussion by Dr. Conrad Berens, New York); "Surgical Treatment of Sympathetic Ophthalmia and Its Complications," Dr. John F. Gipner, Rochester; "A Survey of Cases of Sympathetic Ophthalmia Occurring in New York State," Dr. Harold H. Joy, Syracuse; "Treatment of Blood Stream Infections with Gold Sodium Chloride," Dr. Ivan J. Koenig, Buffalo.

Association for Research in Ophthalmology.—Following is a tentative program for the meeting of the Association for Research in Ophthalmology, to be held at Atlantic City, June 11:

Forenoon.—1. Studies on the Retinal Circulation by Direct Microscopy. Dr. Robert K. Lambert, New York. 2. Uveal Sarcoma (Malignant Melanoma): Study Based on Unreported Cases from the Massachusetts Eye and Ear Infirmary. Dr. Theodore L. Terry and Dr. Juanita P. Johns, Boston. 3. Aniseikonia—A Factor in the Function of Vision. Adelbert Ames Jr., Professor of Research in Physiqlogical Optics, Dartmouth Medical School, Hanover, N. H. 4. Measurement (Roentgenometry) of the Anteroposterior Diameter of

the Eyeball in Situ Correlated with Micrometer Measurement Following Enucleation. Dr. Dewey Katz and Dr. Alfred C. Ledoux, Divisions of Ophthalmology and Roentgenology, the University of Chicago.

Afternoon.—1. The Relation of Vitamin A to Anophthalmos in Pigs. Fred Hale, Chief, Swine Division, Texas Agricultural Experiment Station. 2. A Study of the Pneumococcus Group from the Inflamed Conjunctiva and Lacrimal Sac. Dr. S. Hanford McKee, Clinical Professor of Ophthalmology, McGill University, Montreal, Canada. 3. Streptococcic Pseudomembranous Conjunctivitis. Dr. H. C. Kluever, State University of Iowa, Iowa City. 4. Lysozyme Content of Tears. Dr. William M. James, Washington University, St. Louis.

Eye Section of Philadelphia County Medical Society.—At a meeting of the Eye Section of the Philadelphia County Medical Society held on April 4 the following program was presented:

"Superficial Punctate Keratitis," with presentation of a patient. Dr. Aaron Brav.

"An Outline of a Conception of Glaucoma." Dr. Mark J. Schoenberg, New York.

"The Relation of Certain General Conditions to Glaucoma." Dr. Joseph B. Wolffe.

"The Surgical Consideration of Glaucoma," Dr. Charles R. Heed.

Academy of Physical Medicine.—The annual meeting of the Academy of Physical Medicine will be held on June 12 and 13 at the Claridge Hotel, Atlantic City. Further information may be obtained from the secretary-treasurer, Arthur H. Ring, M.D., Arlington, Mass.

Oxford Ophthalmological Congress.—The twenty-fifth annual Oxford Ophthalmological Congress will be held at Oxford from July 4 to 6. Sir Harold Gillies will open a discussion on "Plastic Surgery of the Eyelids and Conjunctival Sac." The Doyne Memorial Lecture will be given by Mr. Malcolm Hepburn. The title of the lecture is "The Rôle Played by the Pigment and Visual Fields in the Diagnosis of Diseases of the Fundus." There will be a short talk on "The Results of Treatment in Retinal Detachment of Various Types" (excluding operative details). Papers by Mr. Tudor Thomas, Dr. Bernard Samuels, Mr. Frederick Ridley and Mr. Arnold Sorsby are expected. The master is Mr. C. G. Russ Wood, and the secretary, Mr. F. A. Anderson, of Shrewsbury.

Ophthalmological Society of the United Kingdom.—The annual congress of the Ophthalmological Society of the United Kingdom was held at the Royal Society of Medicine, 1 Wimpole Street, London, W. 1., on April 4, 5 and 6. Mr. R. Foster Moore, president of the society, presided. A meeting of delegates to the International Organization for the Campaign Against Trachoma was held on the first day of the congress, followed by a public scientific meeting at which addresses were given by delegates from France, Egypt, Japan and Poland.

French Ophthalmological Society.—The forty-eighth congress of the Société française d'ophtalmologie will be held in Paris from May 13

to 18. The chief subjects for discussion will be capsulolenticular extraction of cataract, introduced by Dr. de Saint-Martin of Toulouse, and treatment of detachment of the retina. Further information may be obtained from Dr. René Onfray, 6 Avenue de la Motte-Picquet.

GENERAL NEWS

Report on Roentgenology in Ophthalmology.—The Société française d'ophthalmologie has requested Mr. Edward Hartmann (2 Avenue Ingres, Paris) to give a report on "Radiography in Ophthalmology," with an atlas. Dr. Hartmann would be grateful for abstracts or bibliographic references to any work which has been done on this subject sent by his colleagues in ophthalmology. He would also like to have drawings, charts or roentgenograms for publication in the atlas, with the names of the donors attached. The report will not be limited to roentgenography of the eyeball, but will also include roentgenography of the adjoining structures (the lacrimal passages); of orbital lesions, such as osteomas, and of the neighboring regions with ocular symptoms (sinusitis or intracranial disorders).

Obituary

COLMAN WARD CUTLER, M.D.
1862-1935

Dr. Colman Ward Cutler, who died on March 17, 1935, was born in New London, Conn., on April 23, 1862, the son of Dr. Ward Cutler and the former Mary Williams.

After preparatory work at Russell Collegiate and Commercial Institute in New Haven, Conn., he was graduated from Yale University in 1885 and then attended the College of Physicians and Surgeons in New York, receiving his degree in 1889. After spending two years as an intern in the medical service at St. Luke's Hospital, New York, he went abroad for three years, where he studied under Fuchs in Vienna and Tscherning at the Sorbonne in Paris. It was the latter association no doubt which developed his interest in physiologic optics and the non-surgical aspects of ophthalmology.

Dr. Cutler was closely associated with St. Luke's Hospital, first as attending ophthalmologist and then as consulting ophthalmologist; he was with the New York Eye and Ear Infirmary for many years. At various times he was consulting ophthalmologist at the General Memorial Hospital, Woman's Hospital, the New York City Hospital and St. Mary's Hospital for Children. He was also interested in the work of the Society for the Prevention of Blindness.

Dr. Cutler was a member of the New York Academy of Medicine, the New York County Medical Society, the New York Ophthalmological Society and the American Ophthalmological Society. He was also a member of the Century Club, the Therapeutic Club and the National Institute of Social Sciences.

Dr. Cutler was a careful observer and was acurate and painstaking in his examinations. He attained his eminence through his native ability, his skill and personal charm and his splendid training abroad, and most of all through his conscientious devotion to his profession. He followed the best traditions of medical practice and of the highest ethical standards. He was a highly sensitive person, unaware apparently of his own attainments, and a man of sterling uprightness. His judgment of people was true and kind, and he was never known to make a disparaging criticism about any one. Because of his intense interest and high principles, he did much to raise the standard of ophthalmology. Those who recognized his ability and responsive sympathy will not forget the generous spirit in which he went about his tasks.

ALFRED WIENER.

In his social relations, Dr. Cutler bore himself always with great dignity and suavity. (A colleague has likened him in these qualities to Colonel Newcome.) To the buffets of misfortune he submitted without complaint and with an admirable courage that he may well have owed to the sturdy New England stock from which he sprang. His gentleness of manner did not signify any weakness of character, for he was a man of strong will and of hearty likes and dislikes. He was a constant and serious reader quite outside of his professional interests; he was also a sincere and intelligent lover of good music. His many friends will cherish a happy memory of his strong and sympathetic personality.

FRANCIS ROGERS.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Biochemistry

THE DISTRIBUTION OF FLAVINE IN ANIMAL TISSUES. H. VON EULER and E. ADLER, Ztschr. f. physiol. Chem. **223:** 105, 1934.

The oxidation-reduction action of flavine is extraordinarily great, and this substance, which is either identical with or closely related to vitamin G, runs parallel to the vitamin G content in the various tissues. Among the organs investigated, those richest in flavine were the liver and kidney of cattle and the retina of certain fish (mackerel, cod, eel, perch and whitefish). Per gram of fresh weight, these tissues contained from 0.01 to 0.02 mg., but the retina of cattle and sheep contained only from 0.001 to 0.005 mg. The flavine found was in free dialyzable form. The flavine content of the lens, vitreous, cornea, sclera and choroid was too small to be demonstrable by the method used.

J. E. LEBENSOHN.

Blind

CAN BLINDNESS IN ONE EYE CAUSE PHOTOPHOBIA? W. COMBERG, Ztschr. f. Augenh. **84:** 113 (Sept.) 1934.

Comberg relates a number of instances in which a person with one blind eye and one normal eye suffered so from photophobia that he was handicapped in his work and was obliged to wear dark glasses. This symptom, Comberg points out, does not result from the action of light on the nerve endings of the cornea or the iris of the blind eye—an assumption not infrequently made in textbooks—but from a painful attempt of the sphincter of the iris in the blind eye to contract when light enters the pupil of the normal eye, in other words, from the consensual reaction to light. The contraction of the sphincter of the iris is painful, for in blind eyes there is often a residuum of uveal inflammation. While enucleation relieves the patient of photophobia, the same good effect may be obtained through continued use of a mydriatic in the blind eye.

H. L. BEGLE.

Conjunctiva

A CASE OF PRIMARY ERYSIPELAS OF CONJUNCTIVA BULBI. N. J. SHIMKIN, Brit. J. Ophth. **18:** 583 (Oct.) 1934.

According to Shimkin, only Groenouw has mentioned "that sometimes primary erysipelas starts from the lacrimal ducts or from the conjunctiva." The case observed by Shimkin occurred in a man aged 45 years. The symptoms appeared about thirteen hours after a foreign body lodged in the eye and was washed out by lacrimation. The progress of the condition is given in great detail. The author's summary will suffice:

The symptoms that brought the patient to the doctor were unbearable pains in the left eye and in the corresponding half of the head. A moderate and not extensive edema of the conjunctiva in the region of the rima palpebrarum and punctate hemorrhages on the upper periphery of the scleral conjunctiva quickly increased in size under observation.

The symptoms due to erysipelas of the eye were observed by the author ten hours before the fever and the chill started, and twenty-four hours before the appearance of erysipelas on the face in consequence of an extension of the infection from the conjunctiva of the eyeball to the skin of the eyelids and to the rest of the face.

The primary erysipelatous infection of the scleral conjunctiva was, in all probability, caused by the pathologic Diplococcus *sive* Streptococcus lanceolatus of the type of Pneumococcus Thalamon-Fraenkel that penetrated into the lymphatic vessels of the scleral conjunctiva after its surface had been lacerated by a foreign body.

In examining a patient who complains of unbearable pain in the eye and in the corresponding side of the head, with such insignificant local signs in the conjunctiva of the eyeball, a general practitioner should, in addition to excluding all the appropriate ocular diseases, remember also the possibility of such a rare illness as primary erysipelas of the bulbar conjunctiva, especially when the patient gives a history of previous attacks of erysipelas.

W. ZENTMAYER.

THE PATHOGENESIS AND TREATMENT OF SPRING CATARRH. DEDIMOS, Arch. d'opht. **51:** 277 (May) 1934.

The treatment which Dedimos advocates for spring catarrh consists of subconjunctival injections of a 1:2,000 solution of epinephrine hydrochloride, the amounts being increased from 0.2 to 0.5 cc. This dosage differs from that proposed by Tessier, who reported good results in a series of eighteen cases. Dedimos' results have been good but not so good as those of Tessier. These therapeutic observations are, in his opinion, of interest from the point of view of the pathogenesis of the disease. The relationship to the general condition is discussed especially as regards symptoms which are based on disorders of the vegetative and sympathetic nervous systems. He believes that the disease should be looked on as a condition of vagotonia with ocular predominance excited by the action of the sun's rays. He does not believe that allergy explains many cases. He believes that the favorable results from the subconjunctival injection of epinephrine hydrochloride are due to its local effect on the organic vegetative nervous apparatus.

S. B. MARLOW.

CATAMENIAL CONJUNCTIVITIS: EYE DISORDERS WHICH APPEAR AT THE TIME OF NORMAL MENSTRUATION. J. FRANÇOIS, Arch. d'opht. **51:** 286 (May) 1934.

François reports a case of catamenial conjunctivitis because the occurrence is rare and because he has discovered a method of cure. Three outstanding observations were: the periodic appearance of conjunctival hyperemia for a year before the onset of menstruation, the delayed appearance of the menses at 15½ years, and the regular repeti-

tion of the hyperemia eight days prior to menstruation for seven and one-half years. Treatment was of no avail until he began the injection of 100 rat units of the female sex hormone. This resulted in cure. The various theories to explain the appearances are stated. In view of his personal experience François attributes such conditions to an insufficiency of the sex hormone. Following the presentation of this case he reviews the various eye disorders which may be associated with menstruation. A bibliography is appended.

S. B. MARLOW.

TUBERCULOSIS OF THE BULBAR CONJUNCTIVA. R. BRIDAULT, Bull. Soc. d'opht. de Paris, March 1934, p. 175.

A 53 year old man with pulmonary tuberculosis had a lesion of the conjunctiva near the corneal limbus. The conjunctiva was injected, swollen and discharging. Between the limbus and the cornea a small lump having a tiny white crater in its center was noted. Between the ulceration and the cornea were many fine grayish-gold dots. The submaxillary glands were swollen. The localization described has not been so frequent in the reported cases as the more usual palpebral disease. The reports in the literature indicate that such a condition occurs more commonly in the younger patients. Bridault attributes the condition to a generalized tubercular bacillemia.

L. L. MAYER.

ESSENTIAL ATROPHY OF THE CONJUNCTIVA AND LOSS OF BOTH EYES AFTER ALIMENTARY INTOXICATION. G. WEILL, Bull. Soc. d'opht. de Paris, March 1934, p. 186.

A girl of 21 years, following a meal of warmed-over meat pie with mushrooms, was seized with a severe intestinal intoxication. There were present nausea and profuse diarrhea accompanied by extensive eruptions all over the skin and mucous membranes. Four days after the onset the patient noticed that she was unable to open her eyes. The lids were red and swollen and discharged a seropurulent secretion. The conjunctivae were red and swollen, with an abundance of seropurulent secretion. Near the culdesacs, more especially the lower one, the conjunctiva was drawn out in many bandlike processes. The corneas were cloudy so that neither iris nor pupil was discernible. The ocular motility was good. The general condition was extremely poor. Not only did the skin and mucous membranes show vesicules and papules, but the urine contained sugar, albumin and acetone. The temperature was elevated. The blood culture was sterile. A smear and cultures of the secretion showed Vincent's fusospirilla, enterococci and diplococci. The eyes continued to get worse; after three weeks the corneas perforated, and panophthalmitis resulted. A bad prognosis as to life was given, but after three years the patient recovered. Weill is not satisfied as to whether the condition was one of acute pemphigus or the general polymorphic dermatitis of Duhring and Brocq, or whether it belonged to neither of these classifications. Only two similar cases were found reported in the literature.

L. L. MAYER.

RÔLE OF THE GONOCOCCUS IN THE PURULENT OPHTHALMIAS OF TROPICAL COUNTRIES. V. MORAX, Arch. de oftal. hispano-am. **34:** 345 (July) 1934.

After a brief review of the biologic requirements of the gonococcus and of the usual manner of production of gonococcic infections, Morax refers to the peculiar ocular infections which occur in certain tropical countries (Egypt and Tunis) in the form of seasonal epidemics which are the cause of much blindness and which are apparently transmitted from eye to eye by digital contact (Talbot). The different hypotheses advanced to explain the seasonal outbreaks of the disease—relating to humidity, temperature, sand and overflow of the Nile—do not withstand careful criticism.

In connection with prophylaxis and treatment of the disease, Morax recommends a painstaking and careful inspection of the eyes of persons on the streets and in the dwellings of the poor, in order to initiate the treatment at an early stage and to establish an official isolation of infected persons as soon as possible, combined with an educative campaign among the parents and relatives to counteract local superstitions and prejudicial popular therapeutic measures.

C. E. FINLAY.

Cornea and Sclera

SIMULTANEOUS BILATERAL RODENT ULCER OF THE CORNEA, CURED BY COMBINED CURETTING, THERMOCAUTERY AND MASSIVE CYANIDE SUBCONJUNCTIVAL INJECTION. E. L. JONES, Brit. J. Ophth. **18:** 579 (Oct.) 1934.

The patient was a robust country woman, aged 60 years. Treatment of the ulcer consisted in curetting it and the adjacent sclera forcibly, causing free bleeding and producing a trough as deep almost as the entire thickness of the cornea, and about 3 mm. wide at the middle and about one-fourth the circumference of the ulcer. This area was seared with a postthermocautery, and a massive subconjunctival injection of mercuric cyanide (2 cc. of a 1:1,500 solution) was given immediately, and the eye bandaged. The ulcer healed without further recourse to this treatment.

Subsequently a rodent ulcer developed in the fellow eye and responded to the same treatment. Viosterol was instilled into the eyes daily. Jones states that in extensive ulceration of the cornea from any cause the opacity of the regenerated tissue is much less if massive injections of mercuric cyanide are made than it is if they are omitted.

W. ZENTMAYER.

SPONTANEOUS INTERCALARY STAPHYLOMA. G. MENESTRINA, Lettura oftal. **11:** 13 (Jan.) 1934.

A man of 30 years noted a black nodule at the right upper limbus of the cornea, which increased to the size of a pea. It was diagnosed as a tumor by two oculists, and enucleation was advised. Vision was 3/60 but could be improved to 1/10 by correction of 10 diopters of astigmatism. The nodule, which was formed of ectatic uveal tissue,

was excised, and the edges of the wound were united with corneoscleral sutures and the wound was covered with a conjunctival flap. Healing was uneventful; astigmatism was reduced to 3½ diopters, and with correction of this, vision improved to 3/10. No return of the ectasia was observed after six months. Transillumination of the nodule and its easy reduction by digital pressure facilitated the diagnosis. Sections of the tissue removed showed the existence of adhesion between the iris and sclera at the iris root, presumably due to a focus of uveitis which had given no symptoms.

S. R. GIFFORD.

ISOLATED MELANOSIS OF THE CORNEA EIGHT YEARS AFTER EXCISION OF A SUPERFICIAL MELANOSARCOMA OF THE CORNEAL LIMBUS. ROLF SCHMIDT, Klin. Monatsbl. f. Augenh. **93:** 164 (Aug.) 1934.

The left eye of a man was struck by a hot coal in 1915, but vision remained unchanged until 1922, when a melanosarcoma of the conjunctiva was removed from the temporal portion of the limbus. Prophylactic enucleation was suggested, but was refused by the patient, when fine extensions of pigmented tissue were noted spreading into the sclera. No changes were observed until 1930, eight years after the operation, when the upper temporal periphery of the cornea showed fine, diffused, brown pigmentation. This pigmentation increased, involving nearly the entire periphery of the cornea in July 1932. The globe was removed after three roentgen treatments had seemed to produce an increase of the pigmentation. Histologic examination showed the pigmentation limited to the external layers of the corneal parenchyma. The epithelium was intact; a few isolated strands of tumor cells approached Bowman's membrane near the limbus. The patient's general health had not been affected.

K. L. STOLL.

General

FATALITY RESULTING FROM THE USE OF LASH-LURE ON THE EYEBROW AND EYELASHES. S. B. FORBES AND W. C. BLAKE, J. A. M. A. **103:**1441 (Nov. 10) 1934.

A woman, aged 52, who had considered herself in good health had her right eyebrow "plucked" and the dye "Lash-Lure" applied to the right eyebrow and lashes. Within thirty seconds after the application, the adjacent tissues began to burn and swell, and within a few hours the patient felt ill, and the tissues became greatly swollen. The following day the temperature was 104 F. She was given a mixed streptococcus and staphylococcus vaccine. Eight days after onset she was seen by the authors. The skin over the right side of the forehead and cheek was red but not tense and glistening. The upper lid was covered by a deep slough with much yellowish adherent exudate. The palpebral conjunctiva of the upper and lower lids was completely ulcerated. There was a tenacious exudate. The cornea was hazy, but the epithelium was intact.

A conjunctival culture showed a pure growth of Staphylococcus aureus. There was no time for study of the blood, as the patient died three hours after the first examination by the authors. No autopsy

could be obtained. There is no doubt that the patient's illness was brought about as a violent local and systemic response to the poison.

W. ZENTMAYER.

General Diseases

OCULAR COMPLICATIONS IN PAGET'S DISEASE. J. I. GOUTERMAN, Am. J. Ophth. **17:** 334 (April) 1934.

Gouterman records a case of Paget's disease, giving the observations on the eyes in detail. He makes the following summary:

"The plausible predisposing causative factors in the case before us, namely, the premature birth, the minor and major exanthematae and the chronic sinusitis, can not stand close scrutiny, since patients of premature birth having had exanthematae and suffering from sinusitis do not always develop Paget's disease.

"The probable etiologic causes—the pulmonary tuberculosis, which developed in an active form at the age of 41, and his rheumatic pains of 10 years' standing, somewhat suggestive of a rheumatic diathesis, can not be dismissed lightly.

"The last-named factors could probably favor the development of a bony change in an individual so disposed, considering the devastating effect of tuberculosis on the entire economy. It is, however, problematic.

"Coppez's explanation of the ocular complications of osteitis deformans may be applicable to this case, but at best it is only hypothetical.

"Syphilis, as an etiologic factor, can not be definitely ruled out, in view of the following factors and in spite of a negative Wassermann: (a) diurnal pains worse at night; (b) four accidental (?) miscarriages in the wife.

"The greater bony involvement of the right socket and its surrounding bony structures, as revealed by the x-ray plates, coupled with more extensive pathologic changes in the right eye, may not be merely coincidental. Its possible causative relationship to the pathology of the right eye must be thought of.

"The interesting case herein reported does not justify a definite conclusion relative to the etiology of Paget's disease. It does, however, contribute to, and confirm some of the ocular manifestations thus far recorded in the literature."

W. S. REESE.

ADULT FILARIA (WUCHERERIA BANCROFTI) IN THE ANTERIOR CHAMBER. R. E. WRIGHT, Brit. J. Ophth. **18:** 646 (Nov.) 1934.

A male Hindu, aged 25 years, residing in Madras, suffered from iritis in the right eye. There were a fine exudate on the pupillary border and flocculent deposits at the bottom of the anterior chamber with fine keratitis punctata. There were fine vitreous opacities, the disk was dull red and was indistinctly seen through the haze, and details of the retina were not clear. Two fairly large hemorrhages were seen just beyond the macular area. About two weeks later a filarial worm was seen moving rapidly in the anterior chamber. It was visible to the

naked eye because of its movements and fluorescent sheen. The biomicroscope showed by the characteristic carriage of the tail that the worm was a male. There were punctate brownish deposits all over the endothelium and fine dustlike floaters in the aqueous.

A black mask was clamped onto the lid margins by means of the speculum. A black papier-mâché dish full of warm saline solution was kept ready, with a suitable pipet apparatus in which to receive the filaria as it made its exit. A small keratome incision was made in the cornea, not more than 2 mm. in length, on the deep aspect. The instrument was quickly withdrawn with the loss of only a trace of aqueous. In spite of its length—about an inch—the filarial worm could hardly have been discovered with the naked eye. It seems unlikely that the worm could have been merely washed out by the aqueous but rather, finding itself near an aperture while momentarily there was a forward movement of fluid, it exercised its instinctive urge to push on.

Seven weeks later vision was 6/6. The disk had the appearance of a subsiding neuritis with commencing atrophy. The peripapillary retina was a dull gray.

W. ZENTMAYER.

AN ATYPICAL CASE OF MIKULICZ' SYNDROME. M. MARQUEZ, Ann. d'ocul. **171:** 641 (Aug.) 1934.

This report deals with a case of an atypical syndrome of Mikulicz in which there existed much swelling of the two lacrimal glands and some swelling of the glands of Krause, of the parotid glands, particularly of those on the left side, and of a part of the submaxillary glands.

Removal of the lacrimal tumors was carried out, and radiotherapy was applied to the operative field as well as to the other glands, not removed.

The histologic study of the material showed lymphoid tissue without a vestige of glandular structure. Marquez enlarges on the anatomopathologic aspect of lymphoid infiltration and on the lesions in obscure parts which produce it or favor its production.

S. H. MCKEE.

OPHTHALMOLOGIC SYMPTOMS OF THE SCHÜLLER-CHRISTIAN SYNDROME. K. OBERHOFF, Arch. f. Augenh. **108:** 464, 1934.

This is a review of cases previously reported, with the addition of three cases seen by Oberhoff.

The Schüller-Christian syndrome is caused by a disturbance in fat metabolism affecting only the bones, chiefly those of the skull. The x-ray picture shows destruction of bone and replacement by lipoid granulation tissue. The lesions may be isolated and of varying size, and in the skull this results in the typical roentgen picture of the map contour. The roof of the orbit is the chief site of the changes. In some of the cases which have come to autopsy, lesions were found in the dural sheaths without corresponding changes in the overlying bone, so that it may be presumed that the bony changes are secondary to the changes in the dura.

The symptoms naturally depend on the anatomic site of the bony lesions. Symptoms may arise which suggest bony tumors primarily,

or by pressure the lesions may give rise to symptoms of increased intracranial pressure.

The most frequent symptom, exophthalmos, is due to the presence of bony lesions in the roof of the orbit, or to the ingrowth of xanthomatous granulation tissue in the orbit.

If there are lesions in the region of the sella turcica, diabetes insipidus and disturbances of pituitary function may occur. Although exophthalmos is nearly always present, it may be absent. Paralyses of the extra-ocular muscles are occasionally seen, and choked disk has been reported.

Treatment is of little avail. A diet low in fat, and preparations of thyroid and pituitary gland have been advised. Insulin is recommended by Sosman, and x-rays seem to cause some retrogression in the bony growths.

F. H. ADLER.

CLINICAL RESEARCH ON THE RELATION OF DISEASES OF THE TEETH AND THE EYE. K. SCHMIDT, Klin. Monatsbl. f. Augenh. **93:** 19 (July) 1934.

Feierlink was the first to point out relations between diseases of the teeth and eyes in 1848. Notwithstanding numerous publications on this topic by Rosenow and others, the German ophthalmologists maintain their skepticism in regard to it. Grüter, in 1929, advised one to take all publications on the dental etiology of diseases of the eye with reserve, stating that they lacked definite proof in every respect. Worthy of consideration as sources of ocular infection are, chiefly, chronic inflammations and infections of the apexes of the teeth which have been found roentgenologically, i. e., granulomas and cysts. Alveolar pyorrhea is irrelevant, according to most authors, and caries need not be considered. Possible danger arises as soon as the pulp becomes infected.

Schmidt presents two tables in which he recorded the ocular disease, the roentgenologically established seat of the foci in the teeth, the treatment and the result. In the nineteen cases of the first table no general pathologic disorders were presented by which to establish the etiologic factors in the ocular diseases. In the twenty cases of the second table general diseases were found, such as hypertony, anemia, latent tuberculosis and mostly tuberculosis of the inactive type. All of these patients except one presented granulomas of the dental roots, which were treated, without any influence on the ocular disease.

On the basis of these tables, Schmidt concludes that chronic inflammation of the dental roots has no influence on the eyes. He maintains that most of the reports are too incomplete for use in comparative studies. A general physical examination is necessary, including especially the lungs, with taking of roentgenograms. In this connection, he refers to Nolte, who described ocular diseases as results of old hematogenous tuberculosis, which he found only by means of most carefully taken roentgenograms. Schmidt, furthermore, broaches the question whether the loss of blood and the decay of tissue resulting from dental treatment are not an equivalent of the nonspecific treatment in the shape of parenteral injections which is resorted to in iridocyclitis and other conditions.

K. L. STOLL.

General Pathology

PATHOLOGIC ANATOMY OF VERNAL CONJUNCTIVITIS. A. TORRES, Arch. de oftal. hispano-am. **34:** 126 (March) 1934.

Torres considers vernal conjunctivitis to be of dermoidal nature. There is an increase in the thickness of the palpebral conjunctiva, with multiplication of the layers of overlying epithelium, as well as a considerable hypertrophy of the corium, with isolated formations and cellular infiltration. Eosinophilia is not as constantly present as is usually held. Scrapings show cellular inclusions such as have been reported in trachoma and in nongonococcic ophthalmia neonatorum. He compares the lesions with those of follicular conjunctivitis and trachoma, phlyctenular infiltrations and Parinaud's conjunctivitis. He was not able to find the bacillus described by Bellinzona and considers that all the evidence points to a nonbacterial etiology of the disease under consideration.

C. E. FINLAY.

THE HISTOPATHOLOGY OF SEVERE SYPHILITIC DISEASES OF THE EYE. K. ZABUGIN, Sovet. vestnik oftal. **5:** 318, 1934.

During the year 1930, among 37,143 patients treated in the Woronesh Eye Clinic there were 138 suffering from recent parenchymatous keratitis. None of the patients having old parenchymatous keratitis showed a leukoma of the cornea. The female sex was predominant, and the age of the majority of patients was below 30 years.

The Wassermann reaction was positive in 80 per cent. The severe forms of keratitis parenchymatosa were observed in those patients who had not undergone regular antisyphilitic treatment. In some cases of severe syphilitic disease the eyes were lost. One such case is reported in detail by Zabugin. It concerned a girl, aged 17, who came to the clinic complaining of severe pain and decrease in the size of the right eyeball; this condition she had observed during the past year. A marked ciliary injection with a grayish mass filling the anterior chamber and a soft slightly atrophic eyeball were found on examination. The Wassermann reaction was 4+, and there were symptoms of generalized syphilis. A gumma of the ciliary body was the provisional diagnosis. Since the eye was becoming more atrophic, and the patient had constant pain, it was enucleated.

A detailed pathologic report is given; there was a gumma of the iris, and the iridocyclitis was secondary to this process. The vitreous was also affected, which led to endophthalmitis with involvement of both the retina and the choroid. The eye was lost because of the gumma of the iris, which could have been prevented by early and proper antisyphilitic treatment.

O. SITCHEVSKA.

Glaucoma

ACUTE GLAUCOMA IN A YOUNG MAN. JACQUEAU, Bull. Soc. d'opht. de Paris, March 1934, p. 213.

Jacqueau reports the case of a young man of 19 who had been previously examined and his eyes found to be normal. In 1928 he had his first eye trouble. All the signs of acute glaucoma occurred in the left eye. Tension in this eye was 40; that in the right eye, 15. Myotics brought

the tension to normal in eight days. Frequent exacerbations followed. Consultations with colleagues during the period of remission prevented operation. Finally in 1930 a large peripheral iridectomy not including the sphincter was done. For the past four years no severe attacks have occurred. The visual acuity and fields have remained normal. All physical examinations have given negative results. Jacqueau inquires: Is this due to disease of the sympathetic nerves? The article includes a discussion.

L. L. MAYER.

IRIDOSCLERECTOMY IN INFANTILE GLAUCOMA: LATE RESULTS. PAUFIQUE, Bull. Soc. d'opht. de Paris, March 1934, p. 216.

An infant, 7 months of age, was treated medically for a unilateral glaucoma for several months. The eye appeared to be increasing in size and was somewhat painful. The tension could not be obtained with an instrument because of the behavior of the child. A Lagrange iridosclerectomy was done under general anesthesia. Perception of light increased to ¼ after three years, and the tension remained normal. Paufique feels that in spite of poor vision and a bad prognosis in this type of case operation is indicated.

L. L. MAYER.

Injuries

INTRAOCULAR FOREIGN BODIES. D. MARSHALL, Am. J. Ophth. **17:** 416 (May) 1934.

Marshall reviews eighty cases of intra-ocular foreign bodies and gives the following summary:

"1. A review is presented of the varying opinions with regard to the proper management of intraocular foreign bodies.

"2. A brief analytical summary is made of 80 cases of intraocular foreign bodies.

"3. Study of the literature, and of a series of cases, supports the contention that ophthalmologists should consider more carefully the disadvantages of hasty extraction of foreign bodies, and should, by more observation and less meddling, avoid strenuous and damaging efforts to extract every foreign body.

"4. The prognosis of a case of intraocular retention of a foreign body is generally poor, and in the individual case cannot be foretold."

W. S. REESE.

THE PROGNOSIS OF PENETRATING WOUNDS OF THE GLOBE. M. DOLLFUS and O. HALBRON, Arch. d'opht. **51:** 365 (June) 1934.

This is a statistical review of 394 cases of penetrating wound of the eye, which constituted 4 per cent of all cases seen between 1925 and 1933. These cases are grouped according to sex, age, site of injury, etc. The various types of wounds are considered especially from the point of view of prognosis. In 31 per cent of the cases the involved eye was enucleated. The highest percentage of the enucleations (50 per cent) occurred in the presence of ruptures, injuries due to copper and unremoved foreign bodies. Wounds made by cutting instruments,

glass and metal were the cause of from 20 to 22 per cent of the enucleations. The lowest percentage of enucleations, 18.4 per cent, was in the group of penetrating foreign bodies removed by the electromagnet.

Of the 394 patients, 17 (1 after cataract extraction) had vision of from 8/10 to 10/10, 33 vision of from 4/10 to 7/10 and 44 vision of from 1/10 to 3/10; 100 had vision of less than 1/10; 54 were blind in the injured eye; 115 underwent a mutilating operation. In 34 cases the visual result was unknown. In 5 cases, 1.2 per cent, sympathetic ophthalmia developed; it did not result in total blindness in 2 cases. In résumé, only 23.3 per cent of the patients recovered useful vision. A quarter retained quantitative vision, and a little more than half completely lost vision in the injured eye.

S. B. MARLOW.

ORBITO-OCULAR LESIONS IN FRACTURES OF THE CRANIUM. R. ARGAÑARAZ and J. SENÁ, Arch. de oftal. de Buenos Aires **8:** 6 (Jan.-Feb.) 1933.

After a review of the mechanical, sensory and motor lesions that can be produced by fractures of the cranium, the authors report three cases: One was a case of monocular blindness following a blow on the right side of the forehead, in which a lesion of the nerve within the optic canal was diagnosed. This was confirmed by operation. No visual benefit resulted. The second case was one of monocular loss of vision with oculomotor paralysis from a fracture involving the orbital apex. A decompression alleviated the general but not the local symptoms. The third case was one of pulsating exophthalmos with injury of the carotid in the cavernous sinus following an automobile accident. Some benefit resulted from periodic compression of the carotid artery and intragluteal injection of gelatin.

C. E. FINLAY.

Lens

A SURGICAL ULTRAVIOLET LAMP AS AN AID TO THE REMOVAL OF DISLOCATED LENSES. H. R. HILDRETH, Am. J. Ophth. **17:** 414 (May) 1934.

Hildreth calls attention to the fact that the crystalline lens has the property of fluorescing under ultraviolet light. He utilizes this in making the lens visible during its extraction in cases in which it has been dislocated. He describes the technic used, and also a lamp that he designed.

W. S. REESE.

THE KNOWN DATA ON THE PROBLEM OF CATARACT. J. NORDMANN. Arch. d'opht. **51:** 76 (Feb.); 203 (March); 297 (April) 1934.

This is a review of all the recent literature in the author's possession relating to the development of opacities of the lens. He presents it because of the increasing difficulty in attempting to keep in touch with the scientific aspect of the problem. The review is divided into sections dealing with the following subjects: anatomy, normal and pathologic; physiology as it relates to nutrition, permeability of the lens capsule, metabolism and experimental cataract; chemistry as concerned with the weights and the inorganic and organic constituents of the lens and with

chemical variations in the body related to the formation of cataract and to the physicochemical properties of the lens and its surroundings. In the last section Nordmann discusses the various theories proposed to explain the pathogenesis of cataract. In each section the more recent work on the subject is reviewed. This paper in itself is an abstraction of the current literature on the subject of cataract. It consequently cannot be satisfactorily treated in the same way. It is a valuable collection of the work of other authors and should be referred to in the original.

S. B. MARLOW.

VITAMIN C IN THE LENS, AQUEOUS AND BLOOD IN NORMAL AND PATHOLOGIC LENS METABOLISM. H. K. MÜLLER and W. BUSCHKE, Arch. f. Augenh. **108:** 368, 1934.

In previous experiments, H. K. Müller (*Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch.* **49:** 168, 1932; *Schweiz. med. Wchnschr.* **63:** 1033, 1933; *Arch. f. Augenh.* **108:** 41, 1933; *Nature,* London **132:** 280, 1933) showed that there was present in the aqueous a reducing substance which apparently had its origin in the normal crystalline lens, for only traces of it could be found in aphakic eyes or in experimentally produced cataracts. Further studies showed that this substance formed a typical oxidation-reduction system, and its physicochemical properties suggested that it was ascorbic acid, i. e., vitamin C.

In the present paper the vitamin C content of the aqueous, lens, vitreous, ciliary body, cerebrospinal fluid and blood of the commoner laboratory animals was determined; and also that of the lens, aqueous and blood of man. The methods are given in full.

The authors' conclusions are as follows:

The aqueous and the lens of most of the experimental animals used contain large quantities of vitamin C. The other parts of the eye also contain vitamin C but in lesser amounts. The individual species of animals, however, show large variations in the vitamin C content of the lens. In rabbits there is a considerable diminution in the amount of vitamin C with increasing age. This is even more striking than the reduction in the amount of glutathione which has previously been shown to occur with increasing age.

The authors believe that the lens gives up large amounts of vitamin C to the aqueous, for it does not occur in such quantities in aphakic eyes, in the blood and in the cerebrospinal fluid. In senile cataract and complicated cataracts of cattle there was much less vitamin C present, which often was associated with a reduction in the amount of vitamin C in the aqueous. In rabbits' lenses which had become cataractous through poisoning with naphthalene, there was less vitamin C.

F. H. ADLER.

THE CHEMISTRY OF CATARACTA NIGRA. H. WAELSCH, Arch. f. Augenh. **108:** 443, 1934.

Two black cataracts were extracted by Elschnig in the capsule from one patient, and their chemical properties were investigated. Theoretical considerations of their chemical properties follow but are too technical to allow abstracting.

F. H. ADLER.

THE REDUCING SUBSTANCES OF THE LENS. F. P. FISCHER, Arch. f. Augenh. **108:** 527, 1934.

The lens contains three water-soluble, oxidation-reduction systems besides the proteins, whose reducing properties are due to their cysteine content. These are glutathione, vitamin C, and the yellow respiratory ferment which is apparently identical with vitamin B_2. The lens also contains vitamin A, which acts as an oxidation catalyzer.

The glutathione is present as a reversible oxidation-reduction system, for one finds both oxidized and reduced glutathione in the lens. The methods for determining the form and localization of glutathione are described.

Vitamin C is present in an irreversible oxidation-reduction system and appears to act, together with glutathione, in freeing energy for the metabolic needs of the lens.

The third system, vitamin B_2, acts in the anaerobic glycolysis of the lens and forms a reversible oxidation-reduction system. In cataractous lenses these oxidation-reduction systems are more or less diminished. Fischer points out that the lack of these systems for freeing energy is not the primary cause of cataract formation, but must be a consequence of the initial changes in permeability which he believes take place.

F. H. ADLER.

Lids

TELANGIECTATIC GRANULOMA—BOTRYOMYCOSIS. A. HAGEDOORN, Brit. J. Ophth. **18:** 561 (Oct.) 1934.

Hagedoorn describes a case of telangiectatic granuloma of the eyelid occurring in a youth aged about 16 years. The latter received a very superficial injury to the lower lid, and after a week a fleshy tumor developed. A few days later, after slight bleeding, the tumor fell off. There was a recurrence. It was then removed surgically, and again recurred, with repeated hemorrhages. It was again removed and did not recur. It occupied the inner third of the lower lid and extended from the free border of the latter to some distance on the cheek. It had the appearance of a granuloma. It was composed of connective tissue with dense masses of cells, in the midst of which numerous small lumens could be detected in many places, and these occurred also in and near the stalk. The peripheral part of the tumor showed an abundance of smaller and larger vessels with scarcely visible walls spread in a tissue resembling an embryonal vitreous body. The tumor had an epithelial covering. In many places it was superficially ulcerated. Only superficially were there staphylococci in great numbers, often arranged in globular masses. This structure recalls the findings in botryomycosis.

Botryomycosis, chiefly met with in horses and apparently only occasionally occurring in man, is characterized by a chronic inflammatory development of granulation tissue and fistulas, causing tumors or swellings of the affected organ under the influence either of a special variety of Staphylococcus or of a special reactive condition of the human tissue. Only one case of botryomycosis of the eyelid is reported in the literature, but a few cases involving other regions of the body are described.

Telangiectatic granuloma is clinically a rather sharply marked off, not uncommon variety of granuloma in man. The etiology is not sufficiently clear. A personal case involving the eyelid is described, and instances in the literature are collected.

The article is illustrated.

W. ZENTMAYER.

THE X-RAY TREATMENT OF BLEPHARITIS. NESSUM ABOU SAIF, Brit. J. Ophth. **18:** 589 (Oct.) 1934.

The author considers blepharitis under two types, blepharitis squamosa and blepharitis ulcerosa. Since conditions of similar pathology and all other conditions in which the hair follicles are affected are suitable for roentgen treatment, a technic was perfected for treating blepharitis with roentgen rays. Success of the treatment consists in a thorough epilation of the cilia by the rays. The eye is made insensitive by the application of a solution of cocaine. A prosthesis is applied, and one lid is held between the limbs of a special forceps having long handles and slightly bent terminals to the ends of which are attached shields of zinc and lead to cut off secondary irradiation. The margins together with the cilia are made to face upward. The rays are applied through a circular diaphragm of lead glass 5 cm. in diameter. The treatment formula is: kilovolts, 120; filter, 1 mm., aluminum; milliamperes, 3; focal skin distance, 30 cm. The epilation dose takes seven minutes. One third of this dose is given every third day. Slight congestion follows after each sitting. The epilation begins about the end of the second week and is complete in three weeks. After this yellow mercuric oxide ointment is applied daily. In all cases, the eyelashes grew in again in about two months. In twenty cases in which this treatment was given, two patients had a relapse and were subsequently cured by a second course of treatment.

W. ZENTMAYER.

Methods of Examination

TABLES FOR ACCURATE RETINAL LOCALIZATION. G. H. STINE, Am. J. Ophth. **17:** 314 (April) 1934.

Stine presents several tables to be used in retinal localization, and gives the following summary: "Using an anatomic eye whose dimensions were the average measurements of normal eyes found in the literature, and the schematic eye of Cowan, the limbus distances along the retinal and scleral arcs and the scleral chord were determined by mathematical calculations and exact graphic construction. No account was taken of the aberrations of the eye, the anatomic asymmetry of the globe, or of variations in corneal curvature or in the depth of the anterior chamber. Corrections for angle alpha were incorporated in the data calculated not only for a retina in its normal position, but also for a retina completely detached in each of the four quadrants.

"The angle and meridian of the line of sight to the retinal lesion once found on the perimeter, the limbus distance in millimeters to the corresponding point on the sclera can be directly read from the tables given. The method is accurate, simple, and quick of clinical application."

W. S. REESE.

PHOTOMETRIC GLASSES. M. TSCHERNING, Arch. d'opht. **51:** 337 (June) 1934.

The author describes a method for making and testing photometric glasses and colored glasses. He advocates the use of such glasses in a logarithmic series because it allows the addition of one glass to another. The unit value for a series of such lenses he designates "photopter" and explains the mathematical considerations in arriving at such a unit.

S. B. MARLOW.

MICRO-INCINERATION OF THE NORMAL EYE. BONAMOUR, Bull. Soc. d'opht. de Paris, June 1934, p. 338.

Bonamour reviews the technic of micro-incineration of the eye first used by Policard in 1921 and presented before the Academy of Sciences in 1923.

The histologic localization of the various fundamental metals is readily obtainable by this method. The simple technic for preparation of the specimen is outlined. Bonamour discusses the various layers and their mineral content. He feels that a study of the normal and abnormal mineral contents of the ocular tissues will contribute new solutions to a number of the problems in pathology.

L. L. MAYER.

THE EFFECTS OF SO-CALLED DOUBLE VITAL STAINING ON THE EYE. C. CORDERO, Arch. di ottal. **41:** 241 (June-Nov.) 1934.

The literature on vital staining and its relation to the reticuloendothelial system is reviewed. Cordero employed 1 per cent lithium-carmine and pyrrole blue in the eyes of rabbits. Instillation and subconjunctival injection proved rather ineffective in staining the deeper structures of the eye. Injections into the anterior chamber resulted in absorption of the dyes by reticulohistiocytes of all the ocular membranes except the retina. This was especially marked in the uveal tract and also in the cornea, where a state of inflammation was set up by the injections. Certain cells showed specific absorption of the pyrrole granules; others, of the carmine, and such cells of the same morphologic type were intimately commingled in each of the membranes.

S. R. GIFFORD.

Neurology

OBSERVATION OF THE LATE MANIFESTATIONS OF EPIDEMIC ENCEPHALITIS. P. DEFOUG, Bull. Soc. d'opht. de Paris, March 1934, p. 196.

A man of 30 years complained of unilateral persistent buzzing for seven months. The diminished hearing of the left side had the characteristics of perception deafness. There was no vertigo. In the space of four days a series of phenomena characteristic of epidemic encephalitis occurred, with a rapidly fatal issue. There was vertigo, accompanied by nausea, and augmented by rapid movements of the head of high frequency. Rest in bed was absolutely necessary. Nystagmus in the lateral gaze and paralysis of convergence and of gaze toward the left were the ocular signs. The photomotor reflexes were lost, and fibrillary contractures of the muscles of the face, more especially the orbicularis,

were noticeable. It was impossible to make a spinal puncture. Defoug cites the case as one of retarded epidemic encephalitis in which the effect was almost exclusively on the cochlear nervous apparatus.

L. L. MAYER.

OPHTHALMOSYLVIAN SYNDROME. C. ESPÍLDORA LUQUE, Arch. de oftal. hispano-am. **34:** 616 (Nov.) 1934.

Espíldora Luque reports three cases of unilateral blindness accompanied by hemiplegia of the opposite side. In all three cases the onset was sudden, and the blindness, due to obstruction of the central retinal artery, was permanent. In two cases the hemiplegia was transitory. The two cases in which the hemiplegia was of the right side presented a corresponding aphasia. He explains the conditions observed by a simultaneous obstruction of the central retinal and sylvian arteries, the former being permanent in all three cases and the latter in one; in the other two the transitory nature of the sylvian obstruction shows it to have been due to angiospasm.

C. E. FINLAY.

Ocular Muscles

THE OPTIC ANGLE IN RELATION TO STRABISMUS. E. L. ARMSTRONG, Am. J. Ophth. **17:** 291 (April) 1934.

This paper is based on a study of sixty-six fetuses. The angle of the optic nerves was measured in situ after the brain and the roof of the orbit had been removed; that is, the angle formed by two lines arising from the nasal border of the junction of each optic nerve with the posterior surface of the eyeball which meet in the medial line of the head on the anterior margin of the optic chiasm. The author states the following conclusions: "The measurements done in this research show that all the measurements of the eyes change in a constant and regular manner, with the exception of the angle of the optic nerve, which curves rapidly and straightens out during the latter two months prenatally. This would substantiate the theory that the optic angle determines the phorias, the narrow angles developing cases of esophoria and esotropia, and the wide angles developing the cases of exophoria and exotropia. Also it is in these cases of wide angles that myopia develops from the increased intraocular pressure caused by convergence."

W. S. REESE.

THE USE OF THE SUPERIOR OBLIQUE AS AN INTERNAL ROTATOR IN THIRD-NERVE PARALYSIS. L. C. PETER, Am. J. Ophth. **17:** 297 (April) 1934.

In a case of third nerve paralysis of traumatic origin, Peter performed (1) a recession of the external rectus, (2) a tuck of the internal rectus and (3) a transplantation of the superior oblique to the attachment of the internal rectus. He describes the technic of the third procedure, in which the chief interest centers. Finally he did a Hunt-Tansley operation for the ptosis. The result was most satisfactory.

W. S. REESE.

OPHTHALMOPLEGIA ASSOCIATED WITH BONY CHANGES IN THE REGION OF THE SPHENOIDAL FISSURE. C. B. V. TAIT, Brit. J. Ophth. **18:** 532 (Sept.) 1934.

The patient was a girl aged 17 years. She had been in normal health until sharp pain developed in the right eye extending to the right temple. With this, vomiting began. There was complete paralysis of all the extra-ocular muscles. The pupil was semidilated and sluggish. Ptosis was complete. There was some hyperesthesia of the right supra-orbital nerve, while corneal sensation was impaired. The fundi were normal. Ten days later the left eye became similarly affected. Roentgenograms showed extensive erosion in the region of the sphenoid sinus with destruction of the clinoid processes of the sella turcica together with most of its floor; but there was no definite evidence of tumor. The interpretation favored erosion in this region rather than absorption by pressure.

There was a rapid response to roentgen therapy. Four years and six months after the onset the patient was in good health and had recovered completely except for some residual weakness of the external rectus of the right eye.

Clinically the case shows many of the features which characterized the condition described by James Collier (*Proc. Roy. Soc. Med.* 14: 10, 1921) and termed "rheumatic periostitis of the sphenoidal fissure."

W. ZENTMAYER.

THE INCONSTANT RELATIONS BETWEEN DEGREE OF DEVIATION AND OPTICAL-FUNCTIONAL CONDITIONS IN CONCOMITANT STRABISMUS. M. CORRADO, Ann. di ottal. e clin. ocul. **62:** 817 (Oct.) 1934.

Corrado reports his findings in 100 cases of concomitant strabismus and in their light discusses the various theories purporting to explain this condition. Eighty-one per cent of the cases were examples of convergent, 9 per cent of divergent, 5 per cent of sursumvergent and 5 per cent of deorsumvergent strabismus, the latter two conditions being usually associated with one of the lateral deviations. While in general convergent strabismus was associated with hyperopia (30 per cent) or hypermetropic astigmatism (51 per cent) and divergent strabismus with myopia, many exceptions were found. Of the cases of convergent squint, vision was normal in 7 per cent, moderately reduced (better than 1/10) in 54 per cent and markedly reduced (below 1/10) in 29 per cent. For divergent strabismus the corresponding figures were 1, 4 and 5 per cent. One patient with hyperopia and normal vision showed divergent strabismus. The relation between vision and angle of deviation varied greatly. Of the patients with normal vision, 2 per cent showed slight squint (less than 15 degrees), 5 per cent moderate (from 15 to 35 degrees) and 1 per cent marked (over 35 degrees). Of those with vision moderately reduced, 7 per cent showed slight deviation, 25 per cent moderate and 7 per cent great (over 35 degrees). For those with vision markedly reduced, the figures were 12, 25 and 16 per cent. It is seen that many patients with much reduced vision showed a very slight deviation, while a number with normal vision showed very high deviation. The same variation existed in the relation between aniso-

metropia and deviation: 18 per cent of those with slight anisometropia showed marked deviation while only 12 per cent showed slight deviation.

After the use of atropine the angle of deviation remained unchanged in 16 of 37 patients, while it diminished or disappeared in the remainder, the eyes remaining straight in a few cases after the effect of atropine had disappeared.

Various theoretical considerations are discussed which might explain this inconstancy between deviation and opticofunctional conditions. Corrado is inclined to favor the possibility that the macular area may vary in size in different persons so that a greater or less angle of deviation is necessary to exclude the macula from fixation in certain cases, all of which must be assumed to show some defect in the central mechanism governing muscle balance. A bibliography is appended to the article.

S. R. GIFFORD.

Orbit, Eyeball and Accessory Sinuses

A CASE OF NASO-ORBITAL MENINGOCELE. J. SÉDAN and P. COULOMB, Ann. d'ocul. **171:** 804 (Oct.) 1934.

Sédan and Coulomb report this case because of the relative rarity of the condition and because of the absence of anything relating to it in the literature during the last few years.

Van Duyse gave a complete bibliography to 1905 in the "Ophthalmological Encyclopedia" of that date. In the same year Rohmer stressed the difficulty of diagnosis in such cases. In 1906, Parsons and Coates presented a case before the Ophthalmological Society of the United Kingdom and reported later that intervention had proved fatal. Peters reported a case of bilateral encephalomeningocele in 1917. Up to 1915, 60 cases had been reported. Most of the articles are not illustrated with x-ray photographs. The details of the present case are given, and two x-ray pictures demonstrate the condition beautifully.

S. H. MCKEE.

ORBITAL PHLEGMON IN INFANTS AND OSTEOMYELITIS OF DENTAL ANLAGE. P. DEFOUG, Bull. Soc. d'opht. de Paris, March 1934, p. 180.

In two infants, aged 4 and 5 months, the beginning of the sickness was well recognized. There was a slight rise in temperature together with a firm infiltration of the lid, which was pale. Above the alveolar processes of the incisor tooth were two small abscesses, in which staphylococci were found. The process progressed rapidly. It may have been due originally to endogenous infection. It was felt that the infection in one case was due to a perimamillary furunculosis in the mother. Two days after the onset the symptoms were generalized, and there was noticeable exophthalmos. This was relieved by puncture of the lower conjunctival sac. Free pus was found. Following drainage, a sequestrum was removed. The other patient had a similar onset with protrusion of the eyeball, followed by thrombosis of the orbital vessels and death.

L. L. MAYER.

Pharmacology

CLINICAL EXPERIENCES WITH TWO NEW PHARMACEUTIC PREPARATIONS. M. GLEES, Klin. Monatsbl. f. Augenh. **92**: 801 (June) 1934.

A tropa-acid-ester of 3-di-ethylamino-2.2-di-methyl propanol is a mydriatic which affects accommodation to a negligible degree. Experiments made at the University Eye Clinic of Cologne, on the eyes of fifty persons, showed slight burning, lasting about ten seconds, and beginning mydriasis about ten to fifteen minutes after instillation, the accommodation being not affected at this period. Maximal mydriasis was obtained after an average of thirty-five minutes, the diameter of the pupil varying between 5 and 8 mm. The average dilatation of the pupils gave a diameter 3.5 mm. beyond the original diameter. A reaction to light and convergence was present during the period of mydriasis, which lasted for about forty minutes. The normal width of the pupil had recurred after from six to seven hours. The mydriasis disappeared quicker in bright light than in the dark.

In some patients, no impairment of accommodation was observed during the entire period of mydriasis. The average decrease of the range of accommodation amounted to 0.6 diopters, and all of the examined persons could read without trouble, notwithstanding the mydriasis. An increase of the intra-ocular pressure by more than 2 mm. of mercury was not observed. The preparation had not been used in glaucomatous eyes at the time of publication of this paper. Mydriasis could not be obtained with this drug in two persons in whom homatropine failed afterward. Glees' report on the other pharmaceutic preparation, the local anesthetic pantocain (p-n-butylaminobenzoyl-di-methylaminobenzoyl-ethylamino-ethanol hydrochloride) tallies with those of other writers. He used it in from 0.25 to 0.5 per cent solutions with good result.

K. L. STOLL.

Pupil

IRIDOPLEGIA IN BOXERS. J. SÉDAN, Bull. Soc. d'opht. de Paris, February 1934, p. 89.

Five boxers examined by Sédan all showed large, fixed pupils. He believes the condition is due to trauma to the stroma of the iris after many years in the ring. There was no clinical or serologic evidence of syphilis. The slit lamp showed many fine tears in the sphincter of the iris.

L. L. MAYER.

IRREGULAR, FIXED PUPIL AND DISCOLORATION OF THE IRIS IN ZONA OPHTHALMICA. L. GENET, Bull. Soc. d'opht. de Paris, March 1934, p. 234.

In 1925 Genet presented before the Ophthalmic Society of Lyons, France, a 64 year old man with a dilated, fixed pupil, consecutive with zona ophthalmica. Present examination shows a normal right eye unaffected by the herpes, with normal direct and consensual pupillary reactions. Three months after the initial acute attack of herpes, the left pupil was noted as being irregular, and fixed to direct and consensual

light and in convergence. The iris was discolored. Present examination reveals no change. In addition, it was found that the pupil of the left eye dilated more when the patient remained in a darkened room for a short period. Syphilis was ruled out. Genet suggests that the condition may be due entirely to a lesion of the ciliary ganglion or to one of the sympathetic system supplying the iris. L. L. MAYER.

Physiology

THE INFLUENCE OF LIGHT INTENSITY, INDUCTION AND SECONDARY STIMULI ON IRRADIATION. S. W. KRAVKOV and G. S. BILETZKY, Arch. f. Ophth. **132:** 379 (Aug.) 1934.

Irradiation is the phenomenon by which bright areas (the "field") appear magnified against a black background. It is, in other words, a special type of spatial induction. The authors have studied it under the influence of the factors indicated in the title. The amount of irradiation is the apparent enlargement of the "field" over its objective size. If the light intensity of the "field" is comparatively low (of course always greater than that of the black or gray background), the phenomenon of negative irradiation can be observed, namely, the brighter area appears smaller than it actually is. With increasing light intensity, at first a neutral point is reached at which no irradiation is noticeable. From that point on, positive irradiation or apparent enlargement of brighter areas becomes more and more pronounced as the light intensity is increased. The phenomenon of irradiation is caused by the imperfection of retinal images, i. e., by the circles of diffusion. The subjective borderline between field and ground corresponds with the retinal point which receives a certain multiple of the amount of light which falls on the retinal points forming the image of the background.

P. C. KRONFELD.

THE DIFFERENCE SENSITIVITY OF THE EYE UNDER THE INFLUENCE OF SECONDARY VISUAL OR ACOUSTIC STIMULI. S. W. KRAVKOV, Arch. f. Ophth. 132: 421 (Aug.) 1934.

Acoustic stimuli or visual stimuli acting on one eye lower the difference sensitivity of the other eye. The effect of the secondary stimuli lasts longer than the stimuli. P. C. KRONFELD.

Retina and Optic Nerve

AMBLYOPIA AFTER HEMORRHAGE. A. S. BARR, Am. J. Ophth. **17:** 396 (May) 1934.

Barr gives the following summary: "The history is given of a case of marked loss of vision after uterine hemorrhage that accompanied an abortion. A well-marked neuro-retinitis was present in each eye and visual-field studies showed central scotomas to be the cause of the visual loss. One eye gradually improved so that almost normal vision was obtained, but there was very little improvement in the other eye. After four months the fundus of each eye was almost normal in appearance but small central scotomas could still be made out." W. S. REESE.

DETACHMENT AND RUPTURE OF THE RETINA. A. HAGEDOORN, Am. J. Ophth. **17**: 400 (May) 1934.

Hagedoorn concludes from three experiments that in a healthy rabbit's eye a rupture of the retina alone is not followed by a detachment. He produced ruptures by introducing a blunt cannula through a trepanation opening into the vitreous. A syringe was connected with the cannula, and the latter watched ophthalmoscopically, and when it was seen to touch the retina, an aspirating movement with the syringe caused a rupture of the retina without hemorrhage. W. S. REESE.

MEDICOLEGAL TESTIMONY IN DETACHMENT OF THE RETINA FOLLOWING SLIGHT OR INDIRECT TRAUMA. P. JEANDELIZE and R. BAUDOT, Bull. Soc. d'opht. de Paris, March 1934, p. 197.

At the International Congress of Ophthalmology in Madrid, Spain, the authors reported on the rôle of traumatism in the etiology of retinal detachment. By indirect trauma is meant such injuries as a blow or a knock on the head, a fall or a straining. Three cases, cited in detail, were accepted by the court. All the patients were myopic, and it was demonstrated that because of this predisposition the indirect trauma received in the line of work was the inception of the retinal detachment. It was found that the lapse of time after trauma before detachment occurred was extremely variable. The limit given by Davidson is not in accord with the authors' experience. L. L. MAYER.

SOME CAUSES OF DETACHMENT IN PERSONS WHO ARE SYPHILITIC. GAULT, Bull. Soc. d'opht. de Paris, March 1934, p. 201.

Gault reviews the eighty-three cases of detachment observed at the ophthalmic clinic in Nancy, France, during the past two years. Of these, nine are classified as syphilitic. Heine, in his 1924 report, attributed only three of seventy-five cases to syphilis. Of Gault's cases three were associated with strongly positive Wassermann reactions, three with partially positive reactions, and three with a history of syphilitic antecedents. In none of these nine cases was there a history of tuberculous antecedents. Seven were associated with myopia of from 4 to 6 diopters, four with large macular lesions of the choroid and the others with peripheral lesions. In six the detachment was operated on by the Gonin method, with immediately favorable results in four cases, and late reattachment in one case. Antisyphilitic treatment was also given.

L. L. MAYER.

ANGIOID STREAKS OF THE RETINA; THE VARIABILITY OF THE MACULAR LESIONS; THE RELATIONSHIP TO PSEUDOXANTHOMA ELASTICUM OF THE SKIN. BONNET and PAUFIQUE, Bull. Soc. d'opht. de Paris, March 1934, p. 222.

This patient's case was reported in the November issue of the *Archives d'ophtalmologie:* A woman of 37 complained of poor direct vision with the right eye. Beginning with a macular hemorrhage, the lesion changed from day to day in the course of six months. Exudation

at the macula was noted after three months. A month later the entire picture changed. A preretinal hemorrhage occurred in the macular area, and the course of the macular veins was deviated. Finally the picture of angioid streaks occurred, and the authors believe that they have confirmed Grönblad's contention that the angioid streaks represent old hemorrhages. Pseudoxanthoma elasticum was found as a complicating condition.

L. L. MAYER.

MACULAR LESIONS AND HEREDITARY NERVE CONDITIONS. BONNET and COLRAT, Bull. Soc. d'opht. de Paris, June 1934, p. 333.

In a mother and daughter the type of macular degeneration described by Stargardt was found. Familial macular degeneration as a phenomenon of nervous disorder is usually accompanied by psychic disturbances as in the Tay-Sachs and Vogt-Spielmeyer types. In both these patients, the mother, aged 43, and the daughter, aged 23, a neurologic examination revealed spasticity of a cerebellar type. The Wassermann reactions were negative. Lumbar punctures disclosed normal spinal fluid. The macular lesion consisted of many brilliant points, while the surrounding retina showed pigmentary degeneration.

L. L. MAYER.

CHANGES IN THE PAPILLA OF NASAL ORIGIN. BONNET, SARGNON and PAUFIQUE, Bull. Soc. d'opht. de Paris, June 1934, p. 349.

A boy 13 years old complained that with his right eye he had been seeing many black spots in his field of vision for the past eighteen days. Visual acuity was normal. The anterior segment and vitreous were normal. The papilla presented a hazy outline, the veins were dilated, and one could observe a circle of edema adjoining the disk on the nasal side. The temporal segment was normal. The visual fields had the usual extent, but there was some constriction for colors, more especially for green. The x-ray photograph of the head, the urine and the Wassermann test were normal. The right nasal passage showed signs of obstruction. The edema of the fundus increased. Several polyps were removed from the nose, and recovery was immediate.

L. L. MAYER.

SENILE DISCIFORM DEGENERATION OF THE MACULA. P. SOUDAKOFF, Chinese M. J. **48:** 981, 1934.

Three cases of senile disciform degeneration of the macula in women ranging in age between 50 and 81 are described. The ophthalmoscopic findings were essentially the same in all three cases. In each the fovea first became edematous, seemed to bulge forward and became gray. Connective tissue then developed beneath the retina, together with the formation of whitish dots around the macular region. Signs of hypertension were present in the retinal arteries, and sclerosis of the choroidal vessels was evident. Vision was greatly impaired, with a central scotoma present in all three cases. The Wassermann reaction was negative, and no abnormality was found in the blood chemistry.

O. P. PERKINS.

ACUTE RETROBULBAR NEURITIS IN MYELITIS: REPORT OF A CASE. S. P. CHANG, Chinese M. J. **48:** 991, 1934.

A 30 year old man suddenly lost vision in both eyes, without ophthalmoscopic changes and without apparent cause. Complete paralysis and anesthesia of both extremities developed twelve days later. Improvement in vision began twenty-one days after the onset, while the lower extremities remained paralyzed for six weeks. On discharge, five months after the onset, there was a partial optic atrophy, with vision reduced to the counting of fingers and with an almost complete motor and sensory paralysis of both lower extremities. A bibliography is appended.

O. P. PERKINS

STUDIES ON RETINITIS PIGMENTOSA. OHASI, Acta soc. ophth. jap. **38:** 75 (June) 1934.

Ohasi studied a number of cases of typical retinitis pigmentosa, several cases of retinitis pigmentosa without pigment and syphilitic retinitis pigmentosa. He comes to the following conclusions: The ophthalmoscopic findings consist in changes in the optic nerve head and in the retinochoroidal vessels, changes in retinal color, opacities in the retina and pigment spots. It is characteristic for the macula to remain healthy. The changes in the nerve head vary according to the stage: At first, hyperemia or anemia is seen; later, yellow atrophy. The retinal vessels gradually begin to narrow in a centripetal direction and show definite sclerosis, but the sclerotic changes found in patients with general vascular sclerosis are missing. There is at first a permanent vascular spasm, with gradual obliteration of the vessels beginning at the periphery. The arteries are more affected than the veins. The changes in the choroidal vessels can be seen only in advanced stages of sclerosis with changes in the pigment layer. It is difficult to say whether the sclerotic changes begin in the vessels of the retina or in those of the choroid. The vorticose veins show no changes. The change in the retinal color is due to the change in the pigmented epithelium. The change consists in a reduction of the red color of the fundus, disseminate or diffuse dirty brown spots and finally a color resembling tapetum. The sequence of these changes in the retina is typical. The opacities may consist either of small round glistening grayish-white dots or of larger grayish-white superficial spots resembling bone corpuscles. Sometimes about the papilla a halo is to be observed. The pigment is either in the form of rudimentary small dots or large spots like bone corpuscles. The fundus changes appear first in a zone between the equator and the posterior pole (first girdle of degeneration) and then gradually extend both anteriorly and posteriorly. Thereafter the second girdle of degeneration slowly appears in the preequatorial region, and a halo forms about the papilla. In the later stages these two girdles unite, and if the degeneration should extend, the macular region will also become involved. Ohasi believes that retinitis pigmentosa without pigment is the beginning of typical retinitis pigmentosa. Retinitis pigmentosa without pigment has fewer changes than the typical, and lacks the second degenerative girdle.

Syphilitic retinitis pigmentosa is characterized by a peculiar color of the fundus and of the opacities and by pigment spots in the retina. Retinal vasculitis and disseminated foci in the choroid are generally present.

Histologic examination showed that the changes occupied the outer layer of the retina. There were atrophy of the nerve elements originating in the neuro-epithelium, uneven degeneration of the pigment layer, hypertrophy, and emigration of the pigment into the retina. Vascular sclerosis extended toward the center and was more marked in the retina than in the choroid, with extensive gliosis of the retina. It was remarkable that the macular region was preserved so long.

The changes in the disk were caused by atrophy of the nerve fiber layer, with gliosis and anemia. The changes in the retinal color were caused by degenerative changes of the pigment layer and the associated gliosis. This also explains the halo and the opacities in the retina. Most pigment spots were superficially placed in the retina and assumed a peculiar shape, as they usually embraced the vessels. The other pigment spots were caused by changes in the pigment layer and were irregular in shape. The narrowing of the retinal vessels was produced by vascular sclerosis. The visibility of the choroidal vessels was caused by degeneration of the outer layers of the retina and a diminution of the pigment. The first degenerative girdle lying between the normal equatorial and macular regions was closely associated with the distribution of the choroidal vessels. The second degenerative girdle depended on the localization of the vorticose veins. The juxtapapillary halo was produced by choroidal atrophy and sclerosis of Zinn's vascular ring. Typical retinitis pigmentosa has a congenital cause which is closely related to a primary centripetal progressive sclerosis of the retino-choroidal vessels. The author reserves his views on the presence of a congenital disturbance of the neuro-epithelium. A. KNAPP.

Trachoma

TRACHOMA IN THE BRITISH COLONIAL EMPIRE: ITS RELATION TO BLINDNESS; EXISTING MEANS OF RELIEF; MEANS OF PROPHYLAXIS. A. F. MACCALLAN, Brit. J. Ophth. **18:** 625 (Nov.) 1934.

This lengthy, in part statistical, paper does not lend itself to abstracting. It is a report read by the author before the joint meeting of the International Organization Against Trachoma, of which he was Vice-President, and the International Association for the Prevention of Blindness, in Paris, May 14, 1934.

An account is given of the incidence of trachoma and acute conjunctivitis in the various parts of the British Empire.

No universal scheme of prophylaxis in all countries can be recommended. The one method that can and should be applied in all countries is treatment in the schools. It is important to have in being a hospital or clinic where parents and relatives can themselves experience the benefit of treatment.

While rigorous treatment in the schools by specialists is desirable, a valuable effect can be produced by the instillation of drops through

the agency of the schoolmaster or his deputy, always provided that this measure is insisted on, and is not merely a neglected order.

Blindness is a common sequela of trachoma complicated by acute bacterial conjunctivitis.

The cure of trachoma occurs by the laying down of cicatricial tissue. The means of inducing this stage is no better now than it was thirty years ago.

W. ZENTMAYER.

RESEARCHES ON THE ETIOLOGY OF TRACHOMA. F. L. CANDIAN, Ann. di ottal. e clin. ocul. **61:** 890 (Dec.) 1933.

Candian carried out an extensive series of inoculations of the human conjunctiva with material from eyes with active trachoma. As subjects he chose patients who were blind from optic atrophy, chronic glaucoma or phthisis bulbi but who had normal conjunctivae. One direct inoculation was negative. Two inoculations with expressed material kept in glycerin four days and one with material kept in glycerin forty-eight hours were negative. Nineteen inoculations with filtrates were performed by various methods. Small Berkefeld and Chamberland filters were employed, and in some cases material from a number of fresh cases was pooled for the purpose. To some filtrates were added fresh cultures of Bacterium granulosis obtained from the Rockefeller Institute. One patient was inoculated with this culture alone. Though some marked local reactions were produced, these subsided within a week or two, without leaving scars. Inclusion bodies, which are considered by the author as characteristic of true trachoma, were never found in scrapings from the conjunctiva of inoculated patients.

Candian explains his negative results from direct inoculations with fresh material as due to individual immunity to trachoma. The negative results with filtrates and bacteria, however, open the questions whether one specific agent is responsible for the disease and whether constitutional factors may not be responsible.

S. R. GIFFORD.

RECENT PROGRESS IN THE STUDY OF THE ETIOLOGY OF TRACHOMA. F. F. TANG, Chinese M. J. **48:** 839, 1934.

This article is a simple, nontechnical review of much recent work on trachoma, a disease which affects at least one hundred million persons in China alone. The controversial views regarding the significance of the Prowazek-Halberstädter trachoma bodies are briefly mentioned. Some original work of the author confirms the lack of relationship between nutritional defects and trachoma. He was also unable to isolate Bacterium granulosis in 227 cases of untreated early trachoma; nor was he able to produce the disease in human subjects with cultures of this organism as supplied by the Rockefeller Institute for Medical Research, New York City. He has also done work to show that Bact. granulosis is very readily killed by a variety of chemical reagents which seem to have no effect on the causative agent of trachoma.

O. P. PERKINS.

Tumors

TRUE GLIOMA OF THE RETINA. C. DEJEAN, Arch. d'opht. **51: 257** (May) 1934.

Dejean reports the occurrence of a true glioma of the retina in a man aged 67. It was an astrocytoma, made up of typical neuroglial cells. The case is unique of its kind so far as the literature is concerned. A review of the literature is presented, with a discussion of the varying terminology which has been proposed. The three kinds of neuroglial cells, astrocytes, oligodendrocytes and microgliocytes, together with the characteristics of tumors made up of them are briefly described. The author's personal observation is presented, with a brief clinical note and an extended histologic description with photomicrographs. This case confirms the opinions expressed by Sourdille and van Duyse that it is quite possible to encounter retinal gliomas which have the same clinical and histologic characteristics as gliomas of the central nervous system.

S. B. MARLOW.

BEGINNING MELANOTIC TUMOR OF THE CHOROID: VALUE OF TRANSILLUMINATION IN EARLY DIAGNOSIS. BONNET and PAUFIQUE, Bull. Soc. d'opht. de Paris, June 1934, p. 343.

Bonnet and Paufique report the case of a 46 year old man with sudden lowering of vision in the right eye. Retinoscopy showed emmetropia. At the extreme limit of the temporal retina at 9 o'clock was a red plaque surrounded by a flat detachment of the retina. There was no mobility to the lesion nor any evidence of further retinal involvement. With the scleral lamp a very obscure zone was found coinciding with the lesion. Enucleation was advised and was accomplished so as to leave a large portion of the optic nerve connected with the globe. The authors have reported two similar cases previously, and again emphasize the importance of transillumination.

L. L. MAYER.

SYMMETRICAL BILATERAL EPIBULBAR EPITHELIOMA. L. MAZZI, Arch. di ottal. **41: 260** (June-Nov.) 1934.

A man of 57 years had had a small growth removed from the right bulbar conjunctiva. It recurred two months later, and Mazzi was consulted. It had invaded the cornea almost to the pupil. No adenopathy was present, and vision was 5/10. In a symmetrical position near the outer limbus of the left eye, a small nodule was noted which was considered a hypertrophic pinguecula. The tumor of the right eye was removed and proved on section to be a basal cell carcinoma. Five months later a recurrence was present on this eye, and on the left eye was a tumor resembling that originally present on the right eye. Both were removed and showed a picture similar to that of the first tumor. There was no recurrence after four years.

In the literature no record was found of a case in which tumors of this nature were present on both eyes. Mazzi considers it likely that chronic irritation of the conjunctiva in this exposed area was the cause of both tumors, and that one or both may have gone through the stage

of pterygium formation with later malignant change. His result leads him to advise making every effort to save an eye so affected by removal of the tumor, even in the case of recurrence. S. R. GIFFORD.

MIXED TUMOR OF THE ORBIT. C. S. DAMEL, Arch. de oftal. de Buenos Aires **8:** 187 (May) 1933.

This is a clinical and pathologic report of a case of mixed epithelial and connective tissue tumor of the orbit. The tumor was encapsulated. It was myxoid in nature with a fibroid formation in the periphery. Connective tissue bands crossed it in different directions. The epithelial tissue occurred in the form of cell nests and in cylindric formations. Damel reviews the literature and is inclined to the view that tumors of this class originate in the lacrimal gland. C. E. FINLAY.

THE HISTOGENESIS OF NEVI OF THE CONJUNCTIVA. K. OBERHOFF, Arch. f. Augenh. **108:** 194, 1934.

Oberhoff believes that the nevi of the conjunctiva arise from an undifferentiated tissue which is morphologically associated with the walls of the smallest blood vessels. It is unnecessary to assume the intervention of tissue metaplasia in order to account for these tumors.

F. H. ADLER.

AN EPITHELIAL TUMOR OF THE IRIS. R. SCHMIDT, Arch. f. Augenh. **108:** 457, 1934.

A 60 year old patient about to be operated on for cataract was found to have a grayish-brown, knoblike swelling in the upper portion of the iris. This was excised when the iridectomy was done and found to be a melanoma arising from the epithelial layer of the iris. Reports of similar tumors in the literature are summarized, and the various possibilities as to the origin of melanomas are discussed.

F. H. ADLER.

Uvea

CONCERNING IRIDODIALYSIS AS A CLINICAL ENTITY: ITS SURGICAL TREATMENT. B. W. KEY, Am. J. Ophth. **17:** 301 (April) 1934

Key reports seven cases and gives the following summary: "In this paper it has been the purpose of the writer to point out three important facts concerning iridodialysis: (1) That iridodialysis is a clinical entity, and not a mere incident in clinical ophthalmology; that this may be true even when it occurs in the presence of other pathologic changes; and that its clinical significance is dependent upon its existence alone or upon conditions produced by other pathologic changes; (2) that the torn iris margin in a case of iridodialysis may become spontaneously reattached or replaced; and (3) that in selected cases large defects may be corrected by a simple operative technic at a minimum risk.

"Spontaneous replacement of the iris may be secured by prompt treatment with atropine instillation and complete rest. This is attributed to three factors: (1) Mydriasis from atropin; (2) blood absorption and organization; and (3) fibrinous exudate incident to the hemorrhage and the reaction to the traumatism."

W. S. REESE.

THE CILIARY PROCESSES: THEIR RELATION TO INTRA-OCULAR SURGERY. A. B. REESE, Am. J. Ophth. **17:** 422 (May) 1934.

Reese calls attention to the likelihood of injuring the ciliary processes in certain operations. He found incarceration of the ciliary processes in six, and excision of the ciliary processes in two, of seven eyes examined. He thinks that this may explain iritis and delayed restoration of the anterior chamber following trephining.

W. S. REESE.

Vitreous

THE CHEMISTRY OF THE VITREOUS. Z. STARY AND R. WINTERNITZ, Arch. f. Augenh. **108:** 433, 1934.

In a previous article (*Arch. f. Augenh.* **107:** 191, 1933) these authors reported a marked difference in the calcium and magnesium content of vitreous humor compared with that in aqueous, blood serum and cerebrospinal fluid. The vitreous was found to have a content of magnesium midway between that of aqueous and that of cerebrospinal fluid, and always higher than that in the blood.

The present paper concerns the percentages of the alkali metals in vitreous as compared with those in the aqueous and blood serum of the same animal. The methods employed were the same as in the previous investigation. Potassium was determined by the method of Kramer and Tisdall, sodium by the method of Barrenscheen and Messinger, chlorine by the Van Slyke method, and phosphorus by the method of Bell-Doisy and Briggs.

No constant difference was found in the potassium and sodium content of vitreous and aqueous. There was a slightly higher concentration of chlorine in the aqueous than in the vitreous. The aqueous contained more than twice the amount of inorganic phosphate contained in the vitreous.

The vitreous contains considerable amounts of organic phosphates, and in this respect differs from the cerebrospinal fluid, which contains only traces of it.

These analyses show that although the vitreous in most respects is similar to the aqueous, its chemical composition is not identical, for there are marked differences in certain electrolytes.

F. H. ADLER.

Therapeutics

THE LOCAL USE OF CAROTENE IN OCULAR DISEASES. S. BALACHOVSKI and F. RATCHEVSKI, Bull. Soc. chim. biol. **16:** 220 (Feb.) 1934.

The authors employed a solution of 1 mg. of carotene in 10 cc. of 5 per cent alcohol. A remarkable analgesic action uniformly occurred

even in cases in which the therapeutic effect was nil. Forty-four patients with corneal disease were treated. In indolent or subacute conditions, such as pannus, ulcers of trachoma or keratitis neuroparalytica, the results were encouraging. In hyperacute conditions, such as pneumococcic ulcer, no benefit resulted. J. E. LEBENSOHN.

THE MALARIAL TREATMENT OF TABETIC OPTIC ATROPHY. H. GASTEIGER, Arch. f. Augenh. **108:** 471, 1934.

A series of patients with tabetic optic atrophy were treated by inoculating them with malaria. In a few of the patients the ocular condition remained stationary for a time, or vision was lost rather slowly, but five of the patients suffered a rapid deterioration which seemed to be due to the treatment. In any event, there was no definite effect of the treatment in staying the tabetic process, and in view of these few cases in which the condition seemed to have been made worse, the author feels that treatment with malaria cannot be recommended for tabetic patients whose optic nerves are damaged. F. H. ADLER.

AUTOHEMOTHERAPY OF TUBERCULOUS IRIDOCYCLITIS AFTER SCHIECK. M. L. KRASNOV, Sovet. vestnik oftal. **5:** 522, 1934.

This treatment consists of introducing the patient's blood into the anterior chamber after the aqueous has been withdrawn by a paracentesis; 0.1 cc. of the blood is sufficient to cover the iris. According to Schieck, it is essential to remove the aqueous containing the so-called *Wandertubercel* of von Szily, also the precipitates, and to have the specific and nonspecific immune bodies of the blood acting directly on the local tuberculous process. He found this method to be harmless and of great therapeutic value.

Krasnov treated nineteen patients. In two patients with tuberculous keratitis and in two with sympathetic ophthalmia no improvement was observed. Indirectly this may serve as an indication that sympathetic ophthalmia is not a tuberculous process. In thirteen of the remaining fifteen cases of tuberculous iridocyclitis there was a complete recovery, and in two, only slight improvement. The injections were given from two to four times at the rate of one every two weeks. In all cases in which the treatment was successful the precipitates on Descemet's membrane and the tubercles absorbed, the posterior synechiae were torn away, and vision improved considerably. Further observations are necessary, also general antituberculous treatment in order to avoid a new infection.

Photographs of the irises of three patients demonstrate well the tubercle before and their absorption after the second or third injection of blood into the anterior chamber. O. SITCHEVSKA.

Toxic Amblyopia

A CASE OF BLINDNESS AFTER N. A. B. FRANK JULER, Brit. M. J. **2:** 809 (Nov. 3) 1934.

A married woman, aged 46, presented a positive Wassermann reaction and was given a course of neoarsphenamine in six doses, commencing with 0.3 Gm. and concluding with 0.9 Gm., together with red mercuric

iodide, and potassium iodide by mouth. After the last injection a cutaneous rash developed. Fifteen days later she rapidly became blind. Perception of light was lost in the left eye, and vision in the right eye was reduced to perception of hand movements. Two days later the disks were white with hazy outlines, and the arteries were narrow, with white lines among them. Glyceryl trinitrate was administered, and a few days later sodium thiosulphate was given intravenously, but without benefit. Two years later there was no perception of light in the left eye and bare perception of light in the right eye. The disks were white with hazy edges. Some of the vessels were small and some were of fair size.

The patient had had no ocular complaint before the blindness. The dimness of vision came on simultaneously in both eyes and rapidly. There was no great edema of the disks, and according to Juler, the whole appearance resembled quinine poisoning. The pathologic site and the mechanism of the lesion are open to discussion. No gumma or other gross lesion was seen, and Juler believes that the arsenic itself affected the optic nerve, the retina or the retinal blood vessels.

He discusses the two arsenic preparations and their toxic effects on the eyes as briefly reported in the literature.

A. KNAPP.

REDUCTION OF VISUAL ADAPTATION AS AN EARLY SYMPTOM OF CHRONIC LEAD POISONING. S. M. KHAYUTIN, Sovet. vestnik oftal. **5**: 561, 1934.

Khayutin examined eighty-six typographical workers, aged from 18 to 45 and found a reduction of visual adaptation in all who were subjects of lead intoxication. This was observed in a number of workers previous to the appearance of general symptoms. Another large group of workers occupied in an industry in which there was no contact with lead were examined for comparison; no reduction of visual adaptation was found.

The test was performed on otherwise healthy eyes with Lasarew's adaptometer. The compositors and stereotypers were affected most, the pressmen little, and the binders had nearly normal visual adaptation. Tuberculosis and anemia were predisposing factors. Careless personal hygiene, indicated by eating in the printing rooms and talking or smoking during work, aided the absorption of lead and caused an early reduction of visual adaptation, as well as general symptoms. Since the test for visual adaptation is simple, Khayutin advocates its importance for prevention of the late, more serious, general symptoms of lead poisoning.

O. SITCHEVSKA.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION ON OPHTHALMOLOGY

Dec. 14, 1934

MR. RANSOM PICKARD, C.B., M.S., *President*

REATTACHMENT OF THE RETINA AFTER DISCISSION OF SOFT CATARACT. MR. MONTAGUE L. HINE.

This is the first case to be recorded of reattachment of the retina in an eye in which discission of soft cataract has been carried out. The patient lost the right eye after operation on a soft cataract when he was 11 years of age. On June 10, 1931, the vision in the left eye was 6/9, and no changes in the retina were visible. On March 27 he complained that a ring appeared in the lower field, and that the sight failed rapidly. When seen by me on April 4 he had a large detachment up and in, with a hole in the retina. I thought it advisable to use the Šafář method, since one can get farther back with the Šafář point, and the operation was done on April 9. Though at first there was some fluid beneath the retina, two months after the operation the entire scotoma up and in had disappeared. The reattachment has held for six months, and the vision is 6/60.

QUININE AMBLYOPIA. MR. E. F. KING.

This married woman took 9 pennyworth of quinine to procure abortion, and three hours after doing so she suddenly became deaf, aphasic and partially blind. During the ensuing few days the blindness increased, but the other symptoms abated. At the stage when the pupils were inactive and there was no perception of light, the retinal vessels showed only slight constriction. By the following day central vision had returned, though one could not demonstrate peripheral fields. A fortnight later, the pupils gave a brisk response to light, though of limited range. At present there is some pallor of the disks, the vessels are a little contracted, and the fields are markedly contracted. Central vision is 6/9 minus.

DISCUSSION

DR. A. J. BALLANTYNE: The case illustrates a point which is not sufficiently noted in textbooks, that at the stage at which vision is most profoundly lost, the appearances of the fundus are practically normal, and that contraction of the retinal vessels occurs at a later stage, when general recovery is taking place. I have seen a number of cases with this order of events, especially in Salonica (Macedonia) during the War, where quinine in heavy doses was much used.

MR. EUGENE WOLFF: I wonder whether in these cases the toxin reaches the retina through the vitreous; if so, does it not explain the vessels being affected after the ganglion cells?

MR. T. HARRISON BUTLER: For four years I treated eyes in Jerusalem, where malaria is endemic and people take enormous doses of quinine. I occasionally saw deafness follow the taking of this drug, but I do not remember having seen amblyopia due to quinine. It seems that there must be some factor required, additional to the quinine, to cause blindness. Or there may be a racial immunity.

MR. E. F. KING: With regard to the effect of quinine, I heard of three men who had been treated for hydrocele, quinine being the sclerosing solution, and each of them was referred for ophthalmic examination and treatment because they complained of misty vision.

CYST OF THE IRIS. MR. D. MAGOR CARDELL.

When I first saw this woman, aged 49, the pupil was crescentic, and the vision was much reduced. After excision of a portion of the anterior wall of the iris, the cyst collapsed, and the vision improved to 6/9. Since then, however, the cyst has been gradually reforming, and I am considering excision of the posterior wall of the cyst.

AVULSION OF THE OPTIC NERVE. MR. D. MAGOR CARDELL.

This boy was knocked down by a push bicycle. The right eye showed normal excursions, but there was a white mass in the vitreous, projecting forward, with strands radiating from it in star formation. No view could be obtained of the optic disk or of the macula. Ten months later, the mass was smaller, disclosing behind its temporal edge a white band of choroidal atrophy stretching between two affected vessels. Further contraction ensued as time passed, and then the fundus showed pigmentary changes round the optic disk. There was some paresis of the right superior rectus. The nerve is damaged beyond repair.

CYST OF THE SCLERA. MR. D. MAGOR CARDELL.

There was no history of either injury or operation, though that does not preclude the possibility of some minor injury, because it often happens that a child who is being nursed injures an eye against a pin or ornament which the mother is wearing in her dress. Such an injury may escape attention, especially if the child is crying at the time. Such an occurrence might explain what is seen here. The cyst has been enlarging rapidly, and last Monday it was encroaching on and splitting the layers of the cornea. The cyst was tapped and thin clear aqueous was withdrawn.

DISCUSSION

MR. EUGENE WOLFF: The cyst will reform if the anterior wall only is excised.

A CASE OF SPONTANEOUS ORBITAL HEMORRHAGE. MR. E. F. KING.

Last evening this woman suddenly had diplopia and pain around the left eye. There is a small ecchymosis of the left lower lid, the eye is proptosed, and there is limitation of movement of the eye upward and outward. There is marked sclerosis of retinal vessels in both eyes.

Jan. 11, 1935

MR. RANSOM PICKARD, C.B., M.S., *President*

CORNEAL GRAFT. MR. B. W. RYCROFT.

A woman aged 48 years had loss of corneal transparency from deep interstitial keratitis with uveitis. She received a corneal graft from a donor who had had a copper wire driven through his upper eyelid and who in consequence had recurrent hemorrhages in the vitreous and loss of useful vision. The graft was the same size in donor and host, and was shelved in the manner recommended by Mr. Tudor Thomas. On the third day the graft could just be seen through the aperture, and it was completely opaque, looking like soft blotting paper. At the end of a week it was clear, the stitch had cut its way out, and the conjunctiva began to retract. A week ago vision was 6/36; with telescopic spectacles, it was 6/24, and with spectacles for reading, Jaeger's test type 4 could be made out.

DISCUSSION

MR. TUDOR THOMAS: In dealing with a small pupil, particularly with a small fixed pupil, it is important to have the graft immediately in front of it. A graft which is clear should be left alone. I have now performed the operation in 20 patients. Of 15 in whom the condition was regarded as suitable for the operation, 10 showed a successful result, the criterion of success being that there was retention of a certain amount of corneal transparency with useful vision.

EXUDATIVE RETINITIS. MR. HAROLD RIDLEY.

The patient is an 8 year old girl. The mother noticed luminosity of the right pupil in certain lights. It is uncertain whether the child has ever seen with the right eye, the vision now being mere perception of light.

The anterior segment of the right eye is free from inflammation. The optic disk is normal. There is a large yellowish-white mass in the macular area, raised 3 disk diameters above the retinal level. A similar but larger patch between the macula and the temporal periphery is raised 7 disk diameters. The retinal vessels in this area show dilatation and tortuosity. Many small glistening patches of subretinal exudate can be seen between the two masses.

In the left eye vision is 6/6 and no abnormality has been found.

TWO CASES OF FAMILIAL MACULAR DEGENERATION IN ADOLESCENCE. MR. HUMPHREY NEAME.

The appearance of the fundus in the two cases is rather different. The girl has somewhat massive blotches of pigment in the macular area, while the boy shows a more granular or peppery appearance in the center of the macula. The amount of damage to vision with comparatively slight signs is illustrated in the case of a 29 year old man who had colloid bodies in the macula, with a central scotoma.

CHOROIDAL SCLEROSIS. MR. ARNOLD SORSBY.

It seems clear that three fairly distinct types of choroidal sclerosis can be recognized:

1. The type described by Edward Nettleship as "central senile areolar choroidal atrophy," an example of which he showed to the Ophthalmoscopic Society (Ophthalmological Society) in 1884. In this type, the lesion takes the form of an oval area which extends from the disk temporally, engulfing the macula. It is bilateral, and a varying degree of atrophy of the choriocapillaris, with conversion of the larger vessels into white lines, is present.

2. The massive peripapillary type. A coronary area of sclerosis radiates from the disk. This kind of lesion is illustrated in Haab's *Atlas der äusseren Erkrankungen des Auges nebst Grundriss ihrer Pathologie und Therapie* (Munich, J. F. Lehmann, 1901).

3. The generalized type, first described by Stanford Morton in 1885. In this type it is not unusual to find a localized area more heavily involved than the rest of the fundus.

Among the possible causes which have been advanced are senile degeneration and syphilis. With regard to the question of age, most of the cases which have been reported have occurred in old persons, but some of the patients showing the condition were young. I shall describe a case in a man aged 23 and another in a girl aged 11. With regard to the question of syphilis as a possible cause, the literature contains record of only three cases in which there was any suggestion of syphilis. It is possible to say with reason that in these cases the condition may have been atypical retinitis pigmentosa. A familial factor has been suggested, but not proved, in the cases of Cuperus (for the peripapillary type), of Guglianetti and of Wilmer (for the generalized type). The significance of this factor is emphasized in the two familial groups shown tonight, the condition in one being of the areolar type, and that in the other, of the generalized variety. In three of nine cases of choroidal sclerosis investigated, extensive cardiovascular disturbances were shown.

DISCUSSION

MR. RANSOM PICKARD: I remember many cases of retinitis pigmentosa which, in the final stage, showed sclerosis of the choroid, but did not show it in the early stage. On the other hand, I have seen advanced cases in which there was apparent sclerosis of the choroid, but in which the pigment was so typical that one could not refuse to believe that there was at least an element of retinitis pigmentosa.

MR. EUGENE WOLFF: It is my feeling that if sections were cut from the involved choroid, obliteration of the choriocapillaris would be found. In no other way can one explain the appearance of the retina, the presence of retinal pigment and the visibility of the choroidal vessels.

MR. ARNOLD SORSBY: The question of retinitis pigmentosa is difficult to answer, and the subject is too large to be dealt with properly in an incidental discussion. With regard to the visual fields, all the patients with generalized choroidal sclerosis had a reduction of vision to mere perception of light. For that reason there has been no question of taking the visual fields. The restricted field may be due to a process of dying, or to an inadequate circulation. I do not think perimetric differences would help to establish that.

GERMAN OPHTHALMOLOGICAL SOCIETY

Fiftieth Annual Meeting, Aug. 6-8, 1934

DR. H. ARRUGA, BARCELONA, SPAIN, *Chairman*

TRANSLATION BY PERCY FRIDENBERG, M.D., NEW YORK

(*Concluded from p. 722*)

Scientific Session, August 8

DR. OLOFF, KIEL, *Presiding*

OPTIC NERVE TUMORS AND NEUROFIBROMATOSIS: TREATMENT. DR. FLEISCHER, Erlangen.

The histologic observations in a case observed clinically by Weigelin of Stuttgart, with observations at autopsy by Schmidtmann of Cannstatt, are reported. A girl, aged 3, had bilateral tumor of the optic nerve and chiasm, and central foci, as well as multiple neurofibromotosis of the peripheral nerves. The father of the patient, too, had neurofibromatosis. The optic nerves showed typical gliomatosis with marked involvement of the intervaginal space and of the papilla. This case is considered of great importance in its relation to the question, still sub judice, of the identity or at least correlation of optic nerve gliomatosis with the manifestations of Recklinghausen's disease, on which point this and other similar cases seem to leave slight, if any, doubt. In future study of patients with such tumors of the optic nerve, special attention must be paid to other, extra-ocular, symptoms of the disease both in the patients and other members of their families and their relatives. On the other hand, patients with peripheral or central neurofibromatosis should be examined carefully for symptoms of optic nerve involvement, and this should include examination of x-ray photographs of the optic foramen and sella turcica and complete studies of the visual fields. These tumors of the optic nerve usually progress and invade the cranial cavity, and in some cases death has ensued later without any relapse of the orbital involvement. Accordingly, the question arises whether the orbital operation is sufficient and whether in intracranial involvement one should not, rather, perform an intracranial operation. This procedure seems logical and one to be considered in the individual case. Radiotherapy, too, must be considered. Further statistical research and follow-up to determine the later course and ultimate outcome of cases in which orbital operations have been done are requisite.

DISCUSSION

DR. GILBERT, Hamburg (after discussing the factor of heredity): I present data on a family in which this condition has been observed in three generations. I myself examined the mother, who has since died. She had atrophy of the optic nerve and a number of the retinal foci described by van der Hoeve. All 4 of her children, 2 sons and 2 daughters, became affected with neurofibromatosis, and the 3 older children showed ocular symptoms. The eldest, not examined by me, died,

blind in both eyes, in a condition of complete mental degeneration, after several years in an insane asylum. The second, a few years younger, is at present also in an institution. The ocular symptoms and those related to the central nervous system are less advanced than in the former case but are progressive. The third patient, a sister, now 27 years of age, shows a marked bilateral deterioration of vision due to disease of the retina and nerve and beginning exophthalmos on the right side. The youngest sister, who became affected when she was 15 years old, has so far shown no ocular symptoms. The family considers this condition to be so fatal and irremediable that the two last-affected sisters had, of their own accord, given up all idea of having offspring, even before there was legislation on this point.

DR. HOFFMANN, Königsberg: The extension of tumor masses backward from the orbit, which they fill more or less completely, into the middle fossa is well seen in x-ray pictures of the base of the skull. (A plate was shown demonstrating this feature in a case of Recklinghausen's disease involving the right middle fossa and the orbit.)

VASCULAR CHANGES IN PHTHISIS BULBI. DR. REICHLING, Berlin (Charité).

Examination of this condition in 19 cases of the most varied duration (from five weeks to fifty years) showed that practically all of the pathologic changes in blood vessels which are recognized histologically by modern pathologists occur in phthisis bulbi. All these lesions cannot be noted in one and the same phthisical globe. However, if one proceeds in the way detailed in this paper, embedding the eyeballs in paraffin and staining specially after the methods of Mallory and van Gieson and for elastic tissue, and so on, the changes present an uninterrupted series involving the various layers of the vessel wall. The only exception is offered by acute necrosing change of the vessel wall, which was not found in a single one of the cases. Based on this extensive material, the following division of lesions of the walls of vessels has been worked out: (1) pathologic changes with a predominant broadening of the zone of the subepithelial basal substance, showing (*a*) histolysis of the basal substance with its sequelae (for example, fibrosis of the intima) and (*b*) proliferation of the basal substance with its sequelae; (2) lesions with predominant broadening of the limiting membrane, showing (*a*) histolysis or hyaline degeneration of the limiting membrane and (*b*) hyperplasia or lamellar elastosis of the limiting membrane; (3) lesions involving mainly the media, showing (*a*) histolysis with rarefaction or complete disintegration of the media and partial or complete disappearance of the musculature of the coat, and (*b*) hypertrophy of the media.

A CASE OF BLUE-ANOMALOUS TRICHROMASIA (TRITANOMALY). DR. OLOFF, Kiel.

A woman of 35, first seen in April 1934, complained of headache and pain in the eyes following a mild contusion of the region of the right eye. The objective findings and adaptation (sic) were normal. Examination of the visual fields showed a marked inversion of the peripheral color limits for blue and green, and these two colors were

invariably confused in naming them. On being questioned, the patient said that for a long time she had been unable to distinguish these two colors correctly; for example, she had confounded the blue and green lights on naval vessels. The starboard, green, lights had always looked blue to her. After looking for a time at blue and green objects in close proximity she became nervous and tired so that she finally lost all impressions of color. In decorating the interior of her new house, seven years previously, she had, as she thought, matched the blue furniture covers with rugs and carpets of the same color. To her great surprise she was told that the floor coverings were the color of green arsenic (*Gift grün*). The 5 year old son of the patient also seems to have a disturbance of blue sensation. Repeated and careful examinations of the color sense were made with the large Nagel anomaloscope, the color top, the spectroscope, color charts and colored wools, which spoke with great probability for a congenital disturbance in the way of Engelking's tritanomaly. Red-green blindness and red-green color anomaly could be definitely excluded. In testing with the usual color charts, those of Cohen and the older editions of Stilling's charts failed completely. The blue-yellow charts (Podesta, Vierling) were interpreted doubtfully or absolutely incorrectly. In tests of finer blue perception with the Stilling-Hertel charts, group 15, the patient recognized only the confusion figures (*Vexierzahlen*) and accordingly showed a lowered perception for blue. Just as in the 3 cases of Engelking, there was here, too, a rise in the specific threshold for blue. In addition to the symptoms of exhaustion of which the patient complained, an increased simultaneous contrast, noted with the anomaloscope, indicated an anomalous trichromatic color perception system. For such examinations the color top is indispensable. The best model is that modified by von Hess and Engelking so that observation takes place through a tubelike arrangement. The case reported is unique in that it was one of a woman with defective color sense for blue. Engelking's patients were all men, as is the general rule in anomalous trichromasia.

DISCUSSION

Dr. Engelking, Cologne: It would be interesting to learn whether in this case there was a shortening at the short wave end of the spectrum, whether there was any weakness in the perception of yellow, and whether it was as marked as the anomaly for blue. This has a certain interest in deciding what place should be assigned to this anomaly as a whole in the general system of disturbances of color perception.

Dr. Oloff, Kiel: The spectroscope and the large Nagel anomaloscope have failed to reveal any shortening of the spectrum at the red end. There might have been a minimal shortening, which could easily have been explained as possibly due to the patient's fatigue just at the time of examination. For this determination the large spectral apparatus of von Helmholtz would have been required. Tests with the color top and with the invariable, peripherally equal perimeter test objects of Engelking and Eckstein showed only that the specific threshold for blue was decidedly higher than normal; there was no rise worth mentioning in the threshold for blue (sic. yellow? P. H. F.).

PHYSIOLOGIC SIGNIFICANCE OF ACUITY OF COLOR PERCEPTION. DR. ENGELKING, Cologne.

A series of variously colored lights, red, orange, yellow, green, blue and white, were reduced to a uniform and equal minimal field luminosity. Tests of visual acuity for these colors of equal minimal field luminosity under good conditions of light adaptation showed values which corresponded exactly for all. With dark adaptation and illumination reduced to the point of inability to distinguish colors, there was an inequality in the visual acuity in the sense of the twilight values of the colors. Especially interesting were the investigations carried out under conditions midway between pure daylight vision and pure twilight vision. Under lowered illumination by which colors could just be distinguished but with which gray and blue appeared definitely more brilliant eccentrically than with central fixation, the figures for visual acuity did not correspond either to those obtained with the minimal field luminosities or to those obtained with the twilight values. On the contrary, visual acuity for blue was markedly low and hardly exceeded that for gray of equal minimal field luminosity, whereas visual acuity for red was decidedly higher. So that in this "mixed" or intermediate adaptation phase an effect appeared which was the direct opposite of the Purkinje phenomenon. The explanation for this unusual and unexpected reaction must be looked for in a special form of cooperation of the two quite differently functioning sets of apparatus: that for light adaptation and that for dark adaptation, respectively. (This was reported and analyzed in detail.) In pure daylight vision, accordingly, the illuminating value of colored lights depends solely on the colorless luminosity of the colors. The nature of the color, as such, is quite immaterial for the visual acuity values and to this extent determination of visual acuity presents a method of heterochromic photometry. In mixed conditions of adaptation, on the contrary, there is a more complicated norm. Here, neither minimal field luminosity nor the twilight value nor even, as might have been surmised, the subjective impression of luminosity that is produced by the color alone is decisive for visual acuity. Other factors play a part which are based on the specific difference between foveal and extrafoveal vision.

DISCUSSION

DR. COMBERG, Rostock: Is it not possible that a complicating factor is presented by fluorescence of the lens which takes place when light waves from the short end of the spectrum are used? This might well play a part in lowering the visual acuity of the dark-adapted eye.

DR. ENGELKING, Cologne: The factor of fluorescence could probably be excluded and hardly came into question for these investigations. For it would have had to be particularly noticeable in the cases studied under pure twilight vision. It was not. On the contrary, the visual acuity under these conditions is in accordance exclusively with the twilight values of the lights employed in testing.

SIGNIFICANCE OF THE BLOOD PICTURE AND OF THE BIOLOGIC LEUKOCYTE CURVE IN OPHTHALMOLOGY. DR. SCHMELZER, Erlangen.

From experience gained in the use of the Schilling white blood count, less stress is to be laid on the determination of a possible diagnostic

significance of the white blood cell picture than on its evaluation in regard to prognosis. For this purpose it is requisite to examine the picture at fixed and definite intervals of time and from the series to construct a so-called biologic white blood cell curve (b. L-K) after Schilling. It was possible with the aid of this procedure to get more or less insight into the course of disease as influenced (sic) by the plotted curves. This was the case in a large series of chronic inflammations of the uveal tract (iridocyclitis, choroiditis) and of the cornea (parenchymatous keratitis), as well as in tabetic atrophy of the optic nerve. The method seems to be of special value in settling the question whether in a case of tuberculous iridocyclitis or choroiditis, tuberculin treatment would offer some prospect of success. It would also give valuable information as to dosage and general management of the therapeutic method. It was shown that there is a variation in the course of the b. L-K observed after injections of 0.1 mg. of TB protein which enables one to recognize definitely the presence of allergy or hypersensitivity of the tissues to tuberculin and what the prospects are for a cure with tuberculin treatment. In the course of fever therapy in cases of tabetic atrophy of the optic nerve and parenchymatous keratitis a marked eosinophilia was noted on several occasions, the significance of which has not yet been fully grasped. But besides this feature there was an extremely varied picture, evidently an indication of the marked influence which treatment by means of hyperpyrexia has on this disease. The fever therapy, which ran its clinical course most favorably, except in 1 case of parenchymatous keratitis, finds no contraindication in the white blood cell picture or at least in the b.L-K.

EXPERIMENTAL AND CLINICAL STUDY OF CORNEAL EPITHELIUM. DR. COMBERG, Rostock.

After a review of the well known experiments of Löhlein with epithelial regeneration, it is demonstrated that the pigmented cells have an affinity for artificially traumatized points and that furthermore the pigment may occasionally emigrate into the dividing line of epithelial growth between lower and upper corneal epithelium. These investigations give a satisfactory explanation of the mode of development of Staehli's corneal line in man. It has been demonstrated that cells containing pigment are regularly present in the neighborhood of the corneal margin of older subjects. It may be assumed that these cells, too, are able to wander into and through the cornea and deposit themselves along the dividing line between the upper and lower epithelial growth, at least if advancing age or intercurrent disease has resulted in the formation of spaces or lacunae in this region. Observation of patients showed that the corneal line is extremely variable as to course and extent. It frequently presents many branchings, varying greatly in their location, and whorl figures are seen which after a time may disappear. It is, to say the least, quite improbable that a mere break in Bowman's membrane is the only essential factor for the genesis of the corneal line, as claimed by Vogt. It seems much more logical to assume that not only the corneal line but other clinical phenomena which run a horizontal course and are seen in the region below the corneal equator can probably be explained as owing their development to the fact that the connecting line between upper and lower corneal epithelial growth is located at this point, and that the region is disposed to various diseases.

CONTRIBUTION TO THE STUDY OF CELLULAR GAS INTERCHANGE (ZELLATMUNG) OF THE CORNEA. DR. RAUH, Giessen.

Investigation of the oxidative processes and capacities of the corneal cells has so far failed to give uniform and conclusive data. A modification of the intracellular indophenol blue synthesis (Nadi reaction) was used to test the reactivity of individual cells of the rat's cornea at various stages of development. It was shown that up to the time at which the lid fissure opens spontaneously all the cells of the cornea, viz., epithelial cells, fixed stroma cells and endothelial cells, give a granular oxydase reaction. After the opening, the cells in the region of the lid fissure show a marked peculiarity. Shortly after this has taken place, eighteen days after birth, there may be a marked reaction, whereas older animals show no granular reaction in this region. Only those portions of the cornea which are covered by the lids continue to show a persistent and invariable granular reaction. If the lid fissure of older animals is kept closed for forty-eight hours, again all the corneal cells show the reaction. The fixed stromal cells and the cells of the endothelium keep this reactivity throughout life. Accordingly, all the cells of the cornea have a definite and demonstrable cell respiration which, to judge by this reaction, is not slight.

LOCALIZATION OF FOCI IN BILATERAL NEUROPARALYTIC KERATITIS. DR. RIEHM, Wurzburg.

If one accepts Behr's conception of the nature and location of the lesion in this disease, it appears that the symptom of bilaterality can be caused by a single intracranial focus only if the process is so situated that it acts simultaneously on both sides and, moreover, just at the point of emergence of the ramus ophthalmicus from the gasserian ganglion, causing an irritation of the trophic sympathetic fibers which at this point pass over from the internal carotid plexus. There are two possibilities: 1. It is conceivable that such a focus must be situated in the median plane and also somewhere in the neighborhood of the stalk of the hypophysis. 2. It is also conceivable that a single unilateral focus situated immediately above the one just mentioned, say in the frontoparietal region, may have a distant action on both roots of the ophthalmic division of the fifth nerve and so on the sympathetic fibers in question. Two cases are reported in which clinical observation required the assumption of one and/or the other of these pathogenic possibilities.

CORNEAL HERPES AND TRAUMA. DR. SCHMIDT, Bonn.

This is a statistical report of 414 cases of herpetic or herpetiform corneal disease; in 230 cases the condition was undoubtedly herpetic; in 184, questionably so. An etiologic connection with trauma could be shown in 1.7 per cent of the former group and in 2.2 per cent of the latter. In the entire series trauma was alleged in only 17 per cent of the cases, and definitely proved to be the exciting cause in not quite 2 per cent. For the recognition of a causal relation between trauma and herpetic inflammation of the cornea one requires (1) that a corneal injury shall have been found by the physician and (2) that the herpetic corneal condition has been observed developing in definite time relation to the proved injury and proceeding from it.

MICROSCOPIC PICTURE OF CORNEAL HERPES. DR. GRÜTER, Marburg.

Two questions were posited: 1. Is there a specific reaction product in the inoculated rabbit cornea? 2. How do the finest nerve endings react to the neurotropic herpes virus? It was found that the microscopic reaction of the cornea to infection by the herpes virus is a regular defense reaction, but that it is in close relation to and intimately connected with the reaction of the normal elementary component parts of the cell, especially that of the Golgi system, just as in vaccinia keratitis which was studied for comparison. This, however, does not imply anything definite in regard to the immunologic relations of one or the other virus. Control tests of immunity conditions failed to show that there was an immunobiologic correspondence between herpes virus and vaccinia keratitis, so that one must still hold to the view originally maintained by Grüter (*Ber. ü. d. Versamml. deutsch. ophth. Gesellsch.* **42:** 162, 1921). The second question was studied by means of a modification of the Dogiel-Arnstein technic for demonstrating the normal topography of the trigeminus in corneal epithelium and the morphology of the finer nerve terminals. This showed, in addition to previous findings, that there was, besides the well known basal plexus, another plexus in the middle epithelial zone of the human cornea and probably in that of animals also. Finally there were a number of plexuses in the uppermost epithelial layers with larger recurrent ramifications of the dividing terminal branches. Nerve end-plates, first described by Dogiel, were found not only in the parenchyma and in the basal epithelium, but in the superficial epithelial layers as well. Herpetic keratitis in rabbits shows the following histologic changes in the nerves: The varicose ganglions undergo a marked inflammatory distention and division. Some vesicles are attached to the parent branch by only a very short, thin stem, so that the finer nerve filaments take on the form of coral twigs. Further study of the course of the inflammatory process in the nerves is not feasible because, as necrosis advances, the nerve tissue takes the stain badly or not at all.

DISCUSSION OF PAPERS READ BY DR. COMBERG, DR. RAUH, DR. RIEHM, DR. SCHMIDT AND DR. GRÜTER

DR. MARCHESANI, Munich: I tried, several years ago, to show indirectly, by another method, that herpes of the cornea has rarely if ever had anything to do with trauma. A statistical study of a large series of cases carried out over a period of several years showed that at times there was a noticeable accumulation of cases of herpes in the months of February and March. During the same season of the corresponding years there was also a marked increase in the incidence of cases of grip. In comparison, the cases of ulcus serpens corneae, a lesion which is beyond doubt related to injury, showed accumulation in the summer months. In those years in which there were no marked epidemics of grip, the incidence of corneal herpes failed to show decided seasonal fluctuations.

DR. KLEIBER, Berlin: In the army medical service the question of the causal interrelation of herpes and trauma is of importance in clearing up the matter of injuries in the service. In the Berlin base hospital an accumulation of cases of herpes has been noted lately in connection

with trauma. Three cases are reported in detail: 2 of disciform keratitis (one immediately following a "switch" with a horse's tail and the other, an iron foreign body) and 1 of dendritic keratitis following a lime burn of the cornea.

DR. KRÜCKMANN, Berlin: I should like to ask Dr. Grüter whether the granules which stud (*garnieren*) the nuclear membrane are to be considered as a physical or as a biologic product.

DR. SCHMIDT, Bonn: It cannot be denied that in exceptional cases there is a definite causal interrelation between herpes and injury. But one must be on one's guard against overestimating the traumatic factor. According to Faverey's statistics and my own, traumatic corneal herpes is undoubtedly very unusual. The special conditions of military service may explain the more frequent observation of cases of traumatic corneal herpes in soldiers.

DR. GRÜTER, Marburg: The tissue change shown, consisting in minute vacuolar structures with dark poles, is not a physical change but the expression of a vital process. In the cells one can plainly see a typical process of division in the finest vacuoles, which shows regularity.

THE INFLUENCE OF GAMMA RAYS ON THE COURSE OF ANAPHYLACTIC AND ALLERGIC PROCESSES IN THE EYE. DR. BLAICKNER, Salzburg.

These studies are based on the observations of von Szily and Arisawa. In the experiments with anaphylaxis the factor of radiant energy was introduced in such a way that the last irradiation was applied immediately prior to the reinjection, and the actual anaphylactic reaction was not affected by the gamma rays. The sum total of the findings was that (1) all the animals, as in the experiments of von Szily and Arisawa, acquired a definite and in most cases a marked degree of anaphylaxis, whether they were sensitized from the cornea or from the circulatory system; (2) the irradiated animals reacted more quickly than those not irradiated, both to the primary corneal injection and to reinjections, and, in regard to the vascular dilatation, more intensely, mostly in the beginning; (3) the inflammatory infiltration of the corneal tissue did not reach the degree of intensity in irradiated animals that it did in the others, and yellowish purulent infiltration around the injection punctures was, almost without exception, conspicuously absent in comparison with the sequelae noted in nonirradiated animals; (4) the restitution *ad integrum* of the injured tissue (transparency of the cornea) took place in a week's less time in the irradiated animals. Conclusions as to the effect of gamma rays on tuberculous disorders of the eye, based on these experiments, follow almost as a matter of course, in view of the decided similarity of the two biologic processes. The beneficial effect of applications of x-rays and radium to tissues presenting many forms of ocular tuberculosis is not to be explained by a nonspecific or a specific desensitization of the tissue. The favorable influence on the course of the disease is rather to be ascribed to the direct action of the rays on tuberculous tissue, to the stimulation of the cells by small doses of the rays and principally to the increased afflux of antibodies from an organism rich in antibodies. Clinical experience with radium treatment of ocular tuberculosis in a large amount of material corroborates these experimental findings. It is predominantly in highly allergic subjects in

the second Ranke stage, particularly those presenting the severe exudative forms, that one notes an especially good reaction to gamma rays. There is a regrettable lacuna in formulating the clinical indications, owing to the fact that one can determine the organism's content of effective antituberculous antibodies only with difficulty and is obliged to depend on an evaluation of the tuberculotoxic allergy. It is known that these two processes or reactions do not necessarily run parallel, and some, at least, of the therapeutic failures may well be explained by this circumstance. As to the indications for one or the other form of treatment from the standpoint of the constitution, in general, I am inclined to say, in contradiction to Werdenberg, that treatment with gamma rays and tuberculin therapy do not cover the same ground; one completes the work of the other.

RECENT EXPERIENCE WITH ULTRAVIOLET THERAPY OF ULCUS SERPENS. DR. HOFFMANN, Königsberg.

The material consists of 400 cases in which treatment with ultraviolet rays was used, in the period from 1922 to 1933, in the University Clinic at Königsberg. The ulcer was small, up to 3 mm. in diameter, in 55; of medium size, up to 6 mm., in 171; large, in 145, and involved the entire cornea in 29. Of the 400 eyes, 13, which were already blind, were enucleated without any treatment by radiation having been given; 4 others were completely destroyed by the ulcer, as treatment could not be carried further, on account of intercurrent systemic disease (acute cardiac failure, pneumonia, gastric ulcer); 8 eyes were enucleated after the ulcer had healed, on account of glaucoma. In 8 eyes the ulcerative process proceeded uninterruptedly. Subtracting the 13 enucleated blind eyes, 5.2 per cent were lost. The small ulcers were all healed, as were almost all those of medium size. About 12 per cent of the eyes with large ulcers were lost. In 170 cured eyes, vision was from 5/4 to 5/10. Fifty patients who had previously had lowered vision, owing to such conditions as pannus and myopia, at least regained their old visual acuity. This series shows clearly the important rôle which age plays in the occurrence and course of ulcus serpens. There were twice as many patients with mild ulcer under 50 as there were over that age. With the severe cases the proportion was reversed. The prognosis became progressively less favorable with advancing age. In the period covered by the report no procedures were employed beyond cauterization, Saemisch section, iontophoresis and applications of zinc sulphate. Since we in the Königsberg clinic have used irradiation in every case of ulcus serpens, only 55 of 958 eyes have been lost. Accordingly we cannot dispense with light treatment.

ACTION OF FRACTIONAL X-RADIATION ON THE EYE. DR. GASTEIGER, Innsbruck.

To determine the reaction of the rabbit's eyes to this treatment, a number of animals were irradiated three times within thirty-five days, each time with 106 roentgens, so that there resulted a total dose of 318 roentgens. Clinically, the irradiated animals presented—in the unexposed eye, too—loss of cilia, conjunctivitis and, after a lapse of six months, typical x-ray cataract. Histologic examination revealed, in addition, complete loss of corneal epithelium in some places and severe

lesions of this tissue in others, manifested in failure to take the stain, disintegration of the nucleus, irregular cell forms, vacuolation and similar changes. In the retina, too, changes were noted affecting the ganglion cells and consisting mainly in formation of vacuoles, shrinkage of the cell, disintegration and clumping of the Nissl flakes (*Schollen*), irregular and deformed nuclei and clumping or complete disintegration of the chromatin substance. The membrana limitans interna was absent, but all the other retinal layers were intact and normal. Fractional x-radiation, accordingly cannot be considered as without danger for the eye, although the damage it causes appears to be less than that to be logically expected from the application of a single dose of the same total intensity. Dr. Gasteiger then presented a case of sarcoma of the superior maxilla which had broken through into the orbit and to which, in the course of one month, irradiation totaling 4,410 roentgens had been applied. Up to the time of death, five months later, the eyes failed to show any clinical sign of damage.

EXPERIMENTAL STUDY OF THE FUNCTIONAL STRUCTURE OF THE SCLERA. (Demonstrations.) DR. KOKOTT, Cologne.

The influence of strains and stresses incidental to special function in determining the architectural and structural modifications of scleral elements and their arrangement as hitherto known has been investigated. The best known example of an analogous functional structure is that of the spongiosa of the human femur, which can be compared closely to the technical construction of a crane. The sclera of the eye of the ox, which was used for these investigations, accepts and deals with the stresses and strains incidental to function. The sum total of tensions is united in the dynamic field, which again is characterized by body form and sources of energy. It was possible to imitate the mechanical conditions presented by these factors and by means of calculation and experiment to present and study a model of the dynamic field (*Kraftfeld*) of the sclera. A comparison of the dynamic field with the fiber structure of the sclera shows that this tissue, quite like the spongy substance of the thigh bone, has a definitely functional structure.

ACCURACY OF DETERMINATION OF VISUAL DEFICIENCY. DR. GRAFF, Rathenow.

Such determinations are influenced by so many accessory factors, many of which have not been standardized, that the figures obtained often mean little. The examiner should use uniform, standard transillumination of the best types, so that they are seen under 500 lux or more, the upper limit being still undetermined but probably lying above 50,000 (? P. H. F.) lux. In this entire area visual acuity is constant. The illumination of the remainder of the examining room is of minor importance, although marked luminosity is advisable, as the eye adapts itself to this condition better and much more quickly than to dark. With these presuppositions the fractional value of the required correcting lens can be determined with the greatest accuracy. Still, if one makes these tests and determinations of values repeatedly on different days and independently one notes variations which make it appear

unnecessary in the case of slight or medium defect of vision to carry accuracy further than the present usual degree of quarter diopters. In such cases the accurate correction of optical errors in the marginal parts of the spectacle lens to be worn by the patient is much more important, as in these cases the spherical and cylindric errors in plane glasses are much greater. Even in cases in which a strictly punctal image cannot be attained by spherical lenses, as in cataract glasses, these errors can be reduced to a minute fraction of the marginal errors incidental to plane lenses.

PHYSIOLOGIC ACTION OF TINTED PROTECTIVE GLASSES. DR. KÜHL, Charlottenburg.

This was determined and registered on a basis of extensive calculations and on that of the data of assured experience and physiologic optics as expressed in the color triangle. Comparison with actual observations of and experiments with subjects wearing protective colored glasses proved the correctness of the results and values which had been reached theoretically and by calculation. It appeared that glass of medium absorption up to 50 per cent, especially those kinds of colored glass tested, brown-gray (Ultrasin C), gray-green (Hallauer), cobalt blue, amethyst and blue-green (Neophan) were approximately equal in protecting against dazzling under conditions of uniform and medium absorption (57 per cent). Gray-green and, to a lesser degree, gray-brown glass, as compared with blue afford greater visual acuity, a higher degree of physiologic luminosity and thus better vision for distance. The color impression and color contrast remain practically unaffected by the color of the glass on account of color adaptation and change of color key (*Umstimmung*). More careful comparison proves that brown-gray glass causes the subjective alterations of the natural colors. The other colors all cause changes which are theoretically undesirable at different points of the color triangle, according to the individual color of the glass. These changes are practically negligible, as they do not exceed from 1 to 2 color tone degrees. Besides this, the blue-gray Neophan glass shows increased saturation in the region extending from deep red to violet-purple, which may be desirable for many purposes, but this increase amounts to hardly 2 degrees of saturation.

RADIODIAGNOSTIC MATERIAL OF THE HALLE CLINIC. DR. VELHAGEN JR., Halle.

X-ray pictures are shown in illustration of cases of orbital injury, the syndrome of the nasal nerve in septal spur, splinter fracture involving the optic canal and foramen in optic nerve atrophy, and various tumors and inflammatory processes in the orbit. Of intrasellar tumors, those are discussed which were accompanied by noticeable polyglandular disturbances or other complications: (1) central scotoma in a patient with empyema of the maxillary antrum and tumor of the hypophysis; (2) hypophyseal tumor with acromegaly, exophthalmic goiter and simple glaucoma; (3) hypophyseal tumor with tapetoretinal degeneration; (4) hypophyseal tumor with diabetes mellitus; (5) genital dystrophy

in a young girl with emmetropia, iridocyclitis, retinal detachment and a markedly small sella, and (6) optic neuritis in a patient with hyperthyroidism and an excessively small sella. A series of cases of secondary wearing away (*Usur*) of the sella are then illustrated in which is presented a marked resemblance to pathologic intrasellar processes. A case of internal hydrocephalus with bitemporal hemianopia is particularly noteworthy. In the category of suprasellar lesions there is shown a tumor presenting evidence of chalky change; also a case of gunshot wound with the projectile lying on the clinoid process and the patient showing clinical symptoms of diabetes insipidus and bitemporal hemianopia. The chapter on pseudoglaucoma is enriched by a number of x-ray pictures showing the shadows of chalky changes, obviously pathologic alterations in the wall of the carotid or of the basal vessels which were projected onto the sella and/or the orbit. Pictures of the fundus of the visual field are shown which are exactly like those seen in cases of true glaucoma. The question of the clinical and pathologic significance of overbridging of the sella (*Sellabrücken*) is then considered en passant, and 2 cases of indefinite (*unklar*) optic nerve atrophy in elderly subjects are presented in which this sellar condition was observed. The significance of pathologic thickening of the clinoid processes is then discussed in the report of 2 cases with changes at the disk resembling glaucoma and enlargement of the blindspots. Cases are also demonstrated of steeple skull and of Crouzon's disease (craniofacial dystrophy with exophthalmos and optic nerve atrophy), and 1 of encephalocele in which a third orbit appears to have hollowed itself out between the other two.

DISCUSSION

DR. HOFFMANN, Königsberg: Attention is called to the diagnostic importance of x-ray pictures taken from special angles and with special technic. Basal views are particularly valuable in the analysis of inflammatory processes or neoplastic formations in the region of the nasopharynx. In hypophyseal exposures a frontonuchal (sic) exposure gives an excellent view of changes around the dorsum of the sella, this structure itself being projected onto the plate through the occipital foramen. Finally, the x-ray picture is shown of a 6 year old child with a gunshot injury of the left eye. There appears what looks like a perforation of the roof of the orbit but is, in fact, an air bubble in the vitreous of the injured eye.

SYMMETRICAL ALEUKEMIC LYMPHADENOSIS. (Demonstrations.) DR. DZIEGELEWSKI, Posen.

A patient had enlarged lymph nodes all over the body. The blood revealed a lymphocytosis of from 30 to 35 per cent. The histologic picture showed at least one feature which has not hitherto been reported. This consisted in a marked hypertrophy of the reticulum cells, especially those of the lymph nodes. This overdevelopment of the reticulo-endothelial system is not to be reckoned as true reticulo-endotheliosis but is rather to be taken, together with the process in the lymph nodes and tumor masses which runs a parallel course, as the expression of a reaction in common to the occult infection.

CHANGES IN THE REGION OF THE OSSEOUS CANAL IN STEEPLE SKULL. DR. BEHR, Hamburg.

Four cases of oxycephaly with optic nerve atrophy have presented convincing anatomic evidence that the nerve lesion is caused by a choking off (*Abdrosselung*) of the optic nerve in the bony canal, not, however, at all owing to an actual stenosis, but rather to a shift in the wall of the canal and a consequent alteration of spatial relations which practically amounts to, or at least results in, the same thing. This is due mainly to an abnormally deep location of the middle fossa with an excessively steep pitch of the roof of the orbit. As a result the roof of the osseous optic canal protrudes backward over its floor. The internal carotid reaches a point below the roof with its fifth turn and thus produces a narrow strait through which the optic nerve has to make its way. The degree of narrowing determines the fate of the nerve. X-ray plates and illustrations of heads with steeple skull are presented which show that this anomalous deep situation of the middle fossa can be noted externally when it manifests itself by a lowering of the external auditory meatus. It is only in such cases that one has an involvement of the optic nerve.

COMMENT

The entire session at Heidelberg ran a very happy course, favored as it was by delightful weather. Participants from foreign lands, who appeared in numbers, will remember the meeting with satisfaction and will have taken home with them the most favorable impressions of a disciplined and internally resolute national-socialistic Germany. The scientific sessions of August 6 and August 7 were adjourned at noon on account of the funeral observances for the deceased Reichs-President, von Hindenburg. To those who shared in the expression of national mourning in the Reichstag and at the burial ceremonies at the Tannenberg National Monument, the community reception in the hall of the Congress will be forever memorable. In the executive session held on August 8, Professor Löhlein of Freiburg was chosen as President, and Sanitätsrat Dr. Jung of Cologne as Vice-President, of the German Ophthalmological Society. The office of permanent secretary of the Society remains as before in the capable and well proved care of Geheimrat Prof. Dr. A. Wagenmann.

Book Reviews

The Biochemistry of the Eye. By Arlington C. Krause, M.A., Ph.D., M.D., F.A.I.C., the Johns Hopkins Hospital. Price, $3.25. Pp. 264, with 17 illustrations and 92 tables. Baltimore: The Johns Hopkins Press, 1934.

In this book Krause summarizes, correlates and critically discusses the results of biochemical research on the eye. The material covers nearly one thousand references scattered over a period of more than one hundred years. The data have been selected from works on physiology, anatomy, pathology, immunology, chemistry and ophthalmology. The material is arranged in eight chapters: "External Secretions and Conjunctiva"; "The Sclera"; "The Cornea"; "The Uveal Tract"; "The Retina"; "The Aqueous Humor"; "The Vitreous Humor," and "The Lens."

Under the subject of external secretions, the tears are the most important topic. The daily quantities secreted under both normal and abnormal conditions are pointed out, and the physical properties and chemical composition are discussed. The secretions of the meibomian and harderian glands have been less extensively investigated. The knowledge concerning the chemistry of the conjunctiva is of very recent date. The data, for which the researches by Krause and Chan are responsible, concern its water content, its total inorganic and lipid constituents and a detailed study of its proteins and their amino-acids.

A more extensive analysis with respect to the inorganic and lipid constituents has been carried out by the same investigators on the sclera, the cornea, the uveal tract and the retina.

The specific chemical nature of visual purple, or rhodopsin, the most important light-sensitive substance in the retina, is practically unknown. Apparently it is in colloidal form or absorbed in colloidal particles. It is believed that visual purple is first bleached to visual yellow, which in turn is bleached to visual white. Considerable space is given to the discussion of photosensitive substances of the cones, including the chemical kinetics of visual purple, the temperature coefficient of the bleaching reaction, the effect of retinal stimulation on visual purple, regeneration of visual purple and vitamin A deficiency. The experimental data on the metabolism of the retina are as yet scanty; it is, however, assumed that it is similar to that of the brain and not of other tissues. Its respiration and anaerobic glycolysis have been found to have the greatest rate of any tissue studied.

Much more definite biochemical knowledge has accumulated with respect to the aqueous and vitreous humors and the crystalline lens.

The data on the aqueous and vitreous humors include, besides a detailed exposition of their chemical compositions, data on their quantities in both man and various animals, the specific gravity, the viscosity, the surface tension (of the aqueous only) and the electrical conductivity and osmotic pressure. The relationship of the aqueous humor to the blood stream is especially emphasized, and the rôle of the exchange

membranes between the two is critically correlated with the principles of the Donnan equilibrium. Biochemical knowledge of the two humors compares favorably with that of the blood.

Greater obstacles have been encountered in a similar study of the lens, on account of its semisolid consistency. However, its protein constituents, together with their basic units, the amino-acids, have been investigated in some detail, and much valuable knowledge has been gained with respect to the rôle of glutathione in the metabolism of the lens.

The book is admirably suited as a reference work for instructors and research workers in the biologic sciences who are interested in the chemical aspect of the physiology and pathology of the eye. The author's style, however, is such that any ophthalmologist with a relatively meager knowledge of chemistry will find the work a delightful and efficient means of enlarging his professional horizon. Well balanced judgment is used throughout in the selection, correlation and interpretation of the various biochemical findings long buried under the maze of technicalities and thus hitherto concealed from the eye of the busy practicing specialist. Numerous tables and more than a dozen diagrams greatly contribute to the lucidity of the presentation.

P. W. SALIT.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217 Faubourg St. Honoré, Paris.
Secretary-General pro tem: Dr. A. Churchill, 66 Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.
Place: Cairo. Time: 1937.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. A. J. Ballantyne, 11, Sandyford Pl., Glasgow, C 3.
Secretary: Dr. J. Ringland Anderson, 108 Collins St., Melbourne.
Place: Melbourne. Time: Sept. 11-13, 1935.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. R. Foster Moore, 53, Harley St., London, W. I.
Secretary: Miss Ida Mann, 7, Wimpole St., London, W. I.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 4-6, 1935.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. J. D. Magor Cardell, 27, Weymouth St., London, W. 1.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.

Chinese Ophthalmological Society in Peiping

President: Dr. P. H. Liu.
Secretary: Dr. H. H. Feng.
Place: Peiping Union Medical College, Peiping.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan.
Place: Cheeloo University School of Medicine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Arthur J. Bedell, 344 State St., Albany, N. Y.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Atlantic City, N. J. Time: June 10-14, 1935.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
President-Elect: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Cincinnati. Time: Sept. 16-20, 1935.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Walter B. Lancaster, Boston.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Hot Springs, Va. Time: June 5-7, 1935.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Frank B. Kistner, Mayer Bldg., Portland, Ore.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Portland, Ore. Time: May 20-23, 1935.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Francis A. Brugman, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Noble Dick, 1115 Terry Ave., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Harry Shearer, 405 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. W. S. Beyer, 1101 Talcott Bldg., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Charles H. Baker, 805 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. J. Hompes, 1307 N St., Lincoln, Nebr.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.
Place: St. Louis. Time: November 1935.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. F. B. Fralich, 203 S. Main St., Ann Arbor, Mich.
Secretary-Treasurer: Dr. A. S. Barr, 201 S. Main St., Ann Arbor, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Claude W. McKee, 229 S. Main St., Greensburg.
Secretary-Treasurer: Dr. C. W. Beals, 41 N. Brady St., DuBois.
Place: Indiana. Time: October 1935.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227, 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Arthur M. Yudkin, 257 Church St., New Haven.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. Herschel C. Crawford, 26 Lindon Ave., N. E., Atlanta.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.
Place: Atlanta. Time: May 7-10, 1935.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Ora G. Brubaker, North Manchester.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.
Place: Indianapolis. Time: April 10, 1935.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. W. Dean, 532, 1st Ave., Council Bluffs.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Council Bluffs. Time: 1935.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. John W. Orr, 503 S. Saginaw St., Flint.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. James A. Reynolds, 78 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. James A. Fisher, 501 Grand Ave., Asbury Park.
Secretary: Dr. William K. Campbell, 96 Third Ave., Long Branch.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION
Chairman: Dr. Webb W. Weeks, 20 E. 53rd St., New York.
Secretary: Dr. John F. Fairbain, 925 Delaware Ave., Buffalo.
Place: Albany. Time: May 13-15, 1935.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. W. R. Winn, 702 First Ave., S., Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.
Place: Minot. Time: June, 1935.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY
Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Josiah E. Smith, 60 Meeting St., Charleston.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Eugene Orr, 706 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. T. E. Fuller, 100 W. Broad St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY
President: Dr. William Stookey, 54 E. South Temple St., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welsh, 711 Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY
President: Dr. Beverley R. Kennon, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. George G. Hankins, Medical Arts Bldg., Newport News.
Place: Fredericksburg. Time: May 4, 1935.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION
President: Dr. W. F. Beckner, 1050 Fifth Ave., Huntington.
Secretary: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Place: Wheeling. Time: May 1935.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Alfred Stahl, 55 Lincoln Park, Newark, N. J.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Herschel Crawford, 478 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. Stacy C. Howell, 384 Peachtree St., N. E., Atlanta, Ga.
Place: Academy of Medicine, 13 Queen's Park. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Lloyd B. Whitham, Mt. Vernon Pl. and Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. John H. Bailey, 855 St. Marks Ave., Brooklyn.
Secretary-Treasurer: Dr. George Freiman, 703 Grand St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. H. H. Glosser, 448 Franklin St., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.
Time: Second Thursday of each month.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. E. V. L. Brown, 122 S. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Thomas D. Allen, 122 S. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. W. C. Davis, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. H. M. Sage, 9 Buttles Ave., Columbus, Ohio.
Place: Athletic Club. Time: First Monday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. W. Mood Knowles, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. L. H. Quinn, 4015 Live Oak St., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. C. Walker, 410 6th Ave., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Secretary-Treasurer: Dr. W. C. Mott, 214 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. T. L. Goodman, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. John H. McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 307 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. R. M. Hargrove, 1202 Esperson Bldg., Houston, Texas.
Secretary: Dr. J. C. Dickson, 1617 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. C. H. McCaskey, 404 Continental Bank Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Raymond E. Teall, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. A. W. McAlester, 2003 Bryant Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Russell T. Uhls, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Kenneth C. Brandenburg, 707 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. F. H. Brandt, 1136 W. Sixth St., Los Angeles.
Secretary-Treasurer: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. E. A. Leggett, 614 Breslin Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. E. Craddock, 604 Breslin Bldg., Louisville, Ky.
Place: Jewish Hospital. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. R. S. Pendexter, 900, 17th St., N. W., Washington.
Secretary: Dr. Lyman B. Tibbets, 1801 Eye St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. J. J. Brook, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6 p. m., second Tuesday of each month.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Secretary: Dr. A. L. Guertin, 410 Sherbrooke St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wesley Wilkerson, Bennie Dillon Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Hale Cullom, Bennie Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Webb William Weeks, 20 E. 53d St., New York.
Secretary: Dr. Guernsey Frey, 121 E. 60th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. John H. Judd, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Robert H. Rasgorshek, 425 Aquila Court, Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Secretary: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward B. Heckel, 500 Penn Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT-LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Samuel M. Cottrell, 116 E. Franklin St., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Laurence Macey, 221 Alexander St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Meyer Wiener, 308 N. 6th St., St. Louis.
Secretary: Dr. W. M. James, Metropolitan Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. Frank Steed, Nix Professional Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Secretary: Dr. Louis F. Morrison, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. John T. Crebbin, 632 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except June, July and December.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Marc Anthony, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. Raymond A. Lower, 1455 Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. D. F. Gillette, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Clarence E. Hill, 160 Bloor St., West, Toronto.
Secretary: Dr. A. Lloyd Morgan, 86 Bloor St., West, Toronto.
Time: First Monday, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. William Thornwall Davis, 927 Farragut Sq., Washington, D. C.
Secretary-Treasurer: Dr. James N. Greear Jr., 1740 M St., N. W., Washington, D. C.
Place: Episcopal Eye and Ear Hospital. Time: 8 p. m., first Thursday in November, January, March and May.